A STUDY ON INCIDENCE OF OPERABLE CARCINOMA BREAST

DISSERTATION SUBMITTED FOR

BRANCH - I M.S (GENERAL SURGERY)

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CHENNAI

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON INCIDENCE OF OPERABLE CARCINOMA BREAST "** submitted by **Dr. G. ANITHA** to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of **M.S Degree Branch – I (General Surgery)** is a bonafide research work carried out by her under direct supervision & guidance.

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DECLARATION

I **Dr. G. ANITHA** declare that, I carried out this work **"A STUDY ON INCIDENCE OF OPERABLE CARCINOMA BREAST"** at the Department of Surgery, Govt. Rajaji Hospital during the period of December 2007 to November 2009. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.S degree examination in General Surgery.

Place : Madurai **Dr. G. ANITHA**

Date :

ACKNOWLEDGEMENT

"Learn to see, learn to hear, learn to feel, and know that by practice alone you can become expert. Medicine is learned by the bedside and not in the class room".

- Sir William Osler

I am greatly indebted to my **Unit Chief and Head of the Department Prof. Dr.M.Gobinath, M.S.,** Department of General Surgery, Government Rajaji Hospital, Madurai for his excellent guidance in conducting this study.

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CONTENTS

ABBREVIATION

BRCA	BREAST CANCER ANTIGEN
FNAC	FINE NEEDLE ASPIRATION CYTOLOGY
DCIS	DUCTAL CARCINOMA IN SITU
LCIS	LOBULAR CARCINOMA IN SITU
EBC	EARLY BREAST CANCER
SLNB	SENTINEL LYMPH NODE BIOPSY
WLE	WIDE LOCAL EXCISION
BCT	BREAST CONSERVATION THERAPY
RT	RADIOTHERAPY
CT	CHEMOTHERAPY
MRM	MODIFIED RADICAL MASTECTOMY
LABC	LOCALLY ADVANCED BREAST CANCER
IBC	INFLAMMATORY BREAST CANCER
CR	COMPLETE RESPONSE
PR	PARTIAL RESPONSE
NR	NO REPONSE
BCS	BREAST CONSERVATION SURGERY
ASCO	AMERICAN SOCIETY OF CLINICAL ONCOLOGY
SEER	SURVEILLANCE EPIDEMIOLOGY AND END RESULTS
HRT	HORMONE REPLACEMENT THERAPY

INTRODUCTION

World wide, Breast cancer is by far the most common cancer amongst women with an incidence rate more than twice that of colorectal cancer and cervical cancer and about three times that of lung cancer. The incidence of breast cancer in India is on the rise and is rapidly becoming the number one cancer in females pushing the cervical cancer to second place.

It is reported that one in 22 women in India is likely to suffer from breast cancer during her lifetime while the figure is definitely more in America where eight being victim of this deadly cancer. The problem with preventing breast cancer is that there is no one cause that can be pinpointed as being the culprit. Ofcourse screening for the presence of BRCA-1, BRCA-2 mutations is available, though it must be admitted of being little use in the Indian context.

The study has been undertaken to emphasise the importance of spreading awareness of the prevalence of this cancer and educating women for self breast examination as a part of early screening step.

Health officials must try & talk about this condition so that women have a say in their own health. Screening procedures like mammography, FNAC & biopsy, need to be widely publicized so as to what exactly they are letting themselves in for.

AIM OF THE STUDY

1. To identify the incidence of operable breast cancer, in Govt. Rajaji Hospital, Madurai Medical College, Madurai.
2. To analyze the clinical staging at the time of admission.
3. To analyze the age wise incidence of carcinoma breast.
4. To improve the awareness regarding early detection of breast cancer by means of multi modality screening procedures as most of the cases in India are locally advanced at the time of presentation.
5. To analyze the preventive measures.
6. To analyze the multimodality treatment options in operable breast cancer.

HISTORICAL ASPECTS

The earliest known medical document to man is that what is known as the Edwin Smith papyrus. Edwin Smith was an Egyptologist who bought fragments of the documents in London in 1862.

In ancient Egypt, the history of breast cancer treatment began with cauterization of tumors found in the breast with an instrument known as the 'firedrill'. Though medical practitioners were able to remove these tumors, they were unable to stop the disease from taking its course. Write the ancient Egyptian doctor of breast cancer, "There is no treatment".

Such was the prevailing wisdom throughout the history of breast cancer. It was not until 17th century in Europe, when the doctors were able to comprehend the nature of the disease and they linked the breast tumors to the lymph glands in axilla.

The pioneering work was done by Sir William Stewart Halsted who began performing complete mastectomies in 1882 and was popular throughout the 20th century until 1970.

In 1952, American Cancer Society created the reach to recovery program, where a group of women helping other women would go and visit patients in hospitals who had just had mastectomies for support and to create awareness.

ANATOMY OF THE BREAST

15 to 20 lobes of tubuloalveolar glandular tissue, fibrous connective tissue that supports it lobe, and the adipose tissue that resides in parenchyma between the lobes. Subcutaneous connective tissue typically does not form a distinctive capsule around breast components, but, rather, surrounds the gland and extends as septa between the lobes and lobules, providing support to the glandular elements. The deep layer of the superficial fascia that lies on the posterior surface of the breast fuses with the deep (pectoral) fascia of the chest wall. A distinct space, the retromammary bursa, can be identified anatomically on the posterior aspect of the breast and resides between the deep layer of the superficial fascia and the deep investing fascia of the pectoralis major and the contiguous muscles of the thoracic wall. The retromammary bursa contributes to the mobility of the breast on the chest wall. Fibrous thickenings of supportive connective tissue interdigitate between the parenchymal tissue of the breast and extend from the deep layer of the superficial fascia to attach to the dermis of the skin. These suspensory structures, known as cooper's ligaments, insert perpendicular to the dermis.

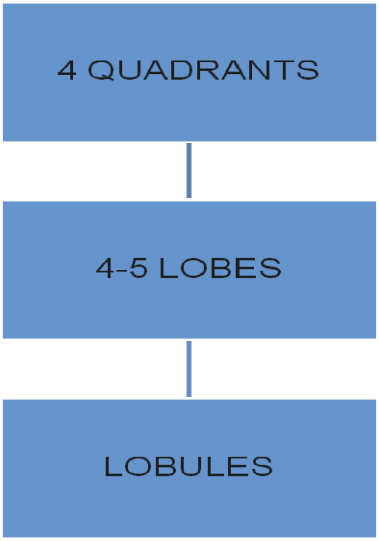
SKIN

- ***THE AREOLA*** : Pigmented , sebaceous glands , Montgomery tubercles

- ***THE NIPPLE*** :

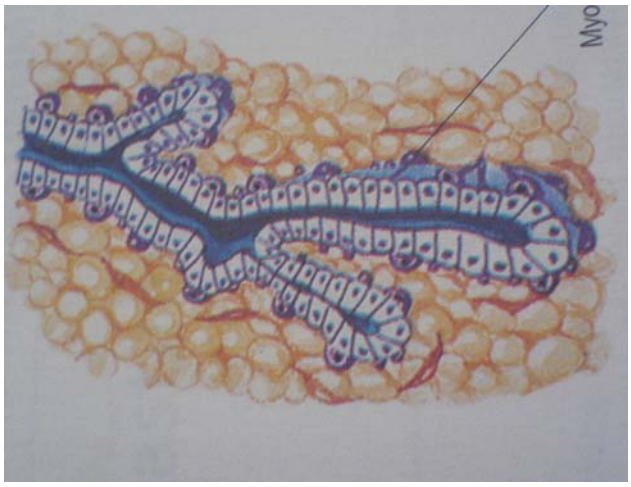
No glands, circumferential muscle fibres and elastic tissue.

STRUCTURAL ANATOMY



MICROSTRUCTURE

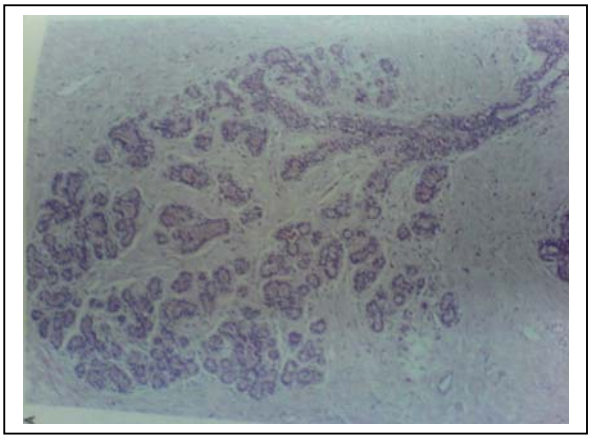
- **Tubulo alveolar glands**
- **Fibrous connective tissue stroma**
- **Interlobular adipose tissue**



STROMA

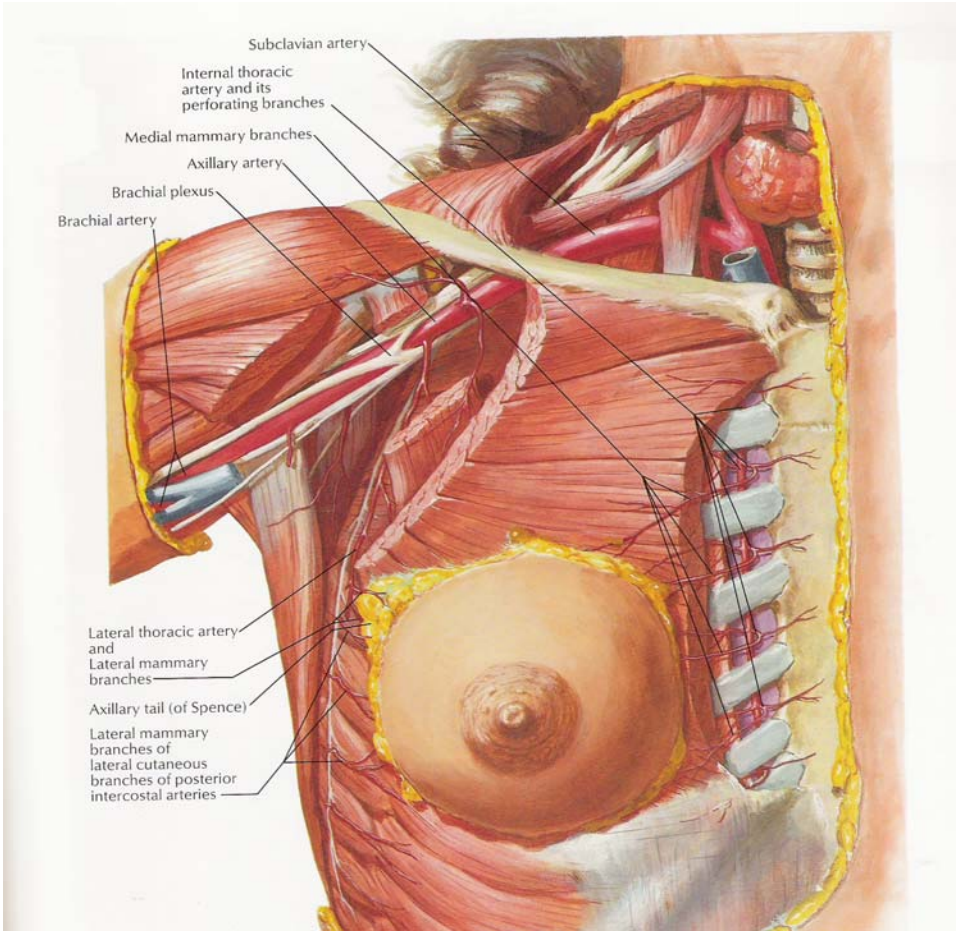
INTRALOBULAR STROMA- Connective tissue stroma has loose texture allowing rapid expansion of secretory tissue during pregnancy

- INTERLOBULAR STROMA- suspensory ligaments (of Astley Cooper) are condensations of fibrous tissue from ducts to dermis, more in the upper quadrants



ARTERIAL SUPPLY

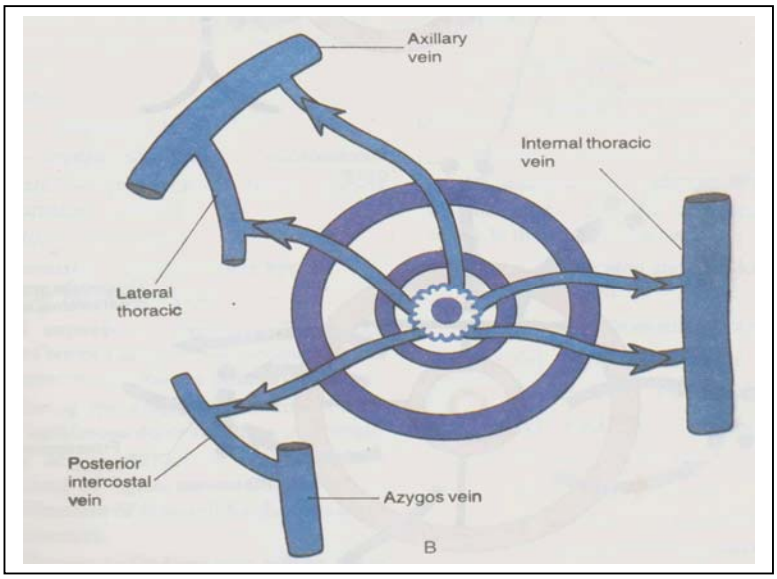
- Lateral thoracic branch of 2nd part of axillary artery
- Medial mammary branches of internal thoracic artery
- Superior thoracic branch of axillary artery
- Lateral branches of 2nd,3rd,4th posterior intercostal arteries



VENOUS DRAINAGE

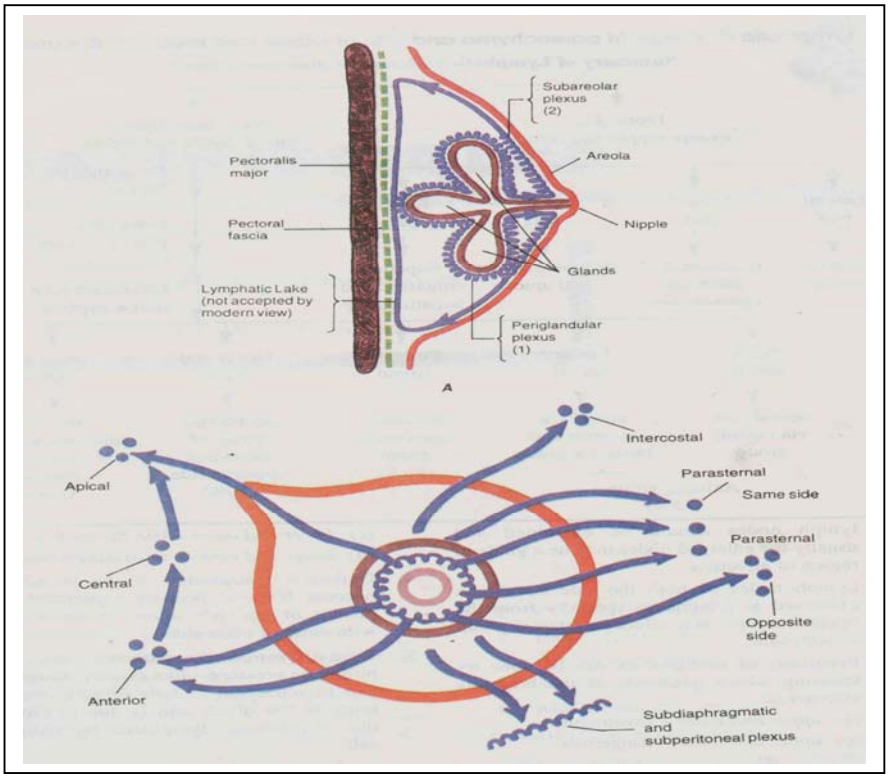
- Circulus venosus - venous plexus deep to the areola
- From this plexus two sets of veins are formed :

1. superficial set- ends in internal thoracic vein
2. deep set - ends in internal thoracic, axillary and posterior intercostal veins



LYMPHATIC DRAINAGE

- AXILLARY NODES- 75 % of lymph drain into axillary nodes
- PARASTERNAL NODES- remaining 25% lymph into parasternal nodes.
- GROUPS – ANATOMISTS - 5
  SURGEONS - 6
    - lateral or axillary vein group
    - external mammary or pectoral or medial group
    - scapular or posterior group
    - central group
    - subclavicular or apical group
    - interpectoral or rotter group



LEVELS OF AXILLARY NODES

LEVEL 1 NODES:

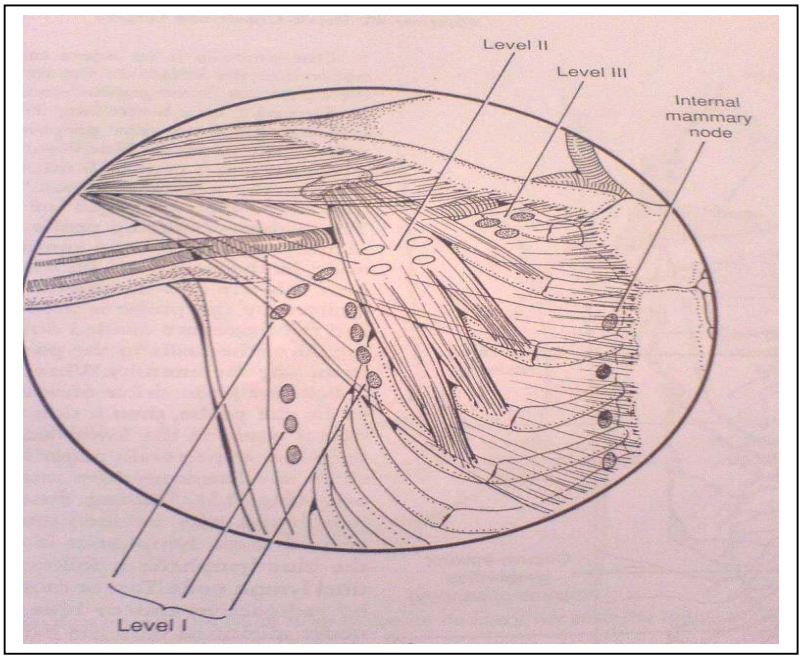
lateral to the lateral border of pectoralis minor muscle external mammary, scapular, axillary vein and central groups

LEVEL 2 NODES:

under the pectoralis minor muscle central, axillary vein group

LEVEL 3 NODES:

subclavicular nodes medial to medial border of pectoralis minor and first rib



ANATOMY OF AXILLARY TENT

- BASE- Axillary fascia
- APEX- aperture that extends into the posterior triangle of neck through CERVICO AXILLARY CANAL
- ANTERIOR WALL – Pectoralis muscles and fascia
- POSTERIOR WALL- Subscapularis , Teresmajor, Lattismus Dorsi.
- LATERAL WALL- Bicipital groove
- MEDIAL WALL- Serratus anterior
- CLAVIPECTORAL FASCIA:
- upper portion –COSTOCOROCOID MEMBRANE pierced by cephalic vein, lateral pectoral nerve, branches of thoraco acromial artery
- Middle portion- pectoralis minor pierced by median pectoral nerve
- Lower portion –CORACO AXILLARY ligament
  HALSTEAD LIGAMENT- medial side of clavicle to the first rib
- AXILLARY ARTERY: 3 PORTIONS identification of the branches of 2^{nd} portion is essential**.**

CARCINOMA BREAST

REVIEW OF LITERATURE

RISK FACTORS

Age

Carcinoma of the breast is extremely rare before the age of 20 years but, thereafter, the incidence steadily rises so that by the age of 90 years nearly 20% of women are affected.

Gender

Female sex carries increased risk. Less than 0.5% of patients with breast cancer are males.

Genetic

It occurs more commonly in women with a family history of breast cancer than in the general population. Breast cancer related to a specific mutation accounts for about 5% of breast cancers. Yet it has far reaching repercussions in terms of counselling and attempted prevention in these women.

Breast cancer syndromes

1. Li-Fraumeni syndrome
2. Cowden's disease
3. Ataxia telangiectasia

Chromosomal Abnormalities

1. BRCA -1 gene mutation – Chromosome 17q
2. BRCA-2 gene mutation – Chromosome 13 q
3. HER-2 mutation (rbB2, transmembrane growth factor)
4. P 53 mutation

Diet

Dietary factors may play a part in its causation, because breast cancer so commonly affects women in the developed world,. There is some evidence that there is a link between diets low in phyto-oestrogens and carcinoma breast. A high intake of alcohol is associated with an increased risk of developing breast cancer.

Endocrine.

Breast cancer is commoner in nulliparous women and breast feeding in particular appears to be protective. Also protective is having a first child at an early age, especially if associated with late menarche and early menopause. It is known that in post menopausal women, breast cancer is more common in the obese. This is thought to be because of an increased conversion of steroid hormones to oestradiol in the body fat. The role of exogenous hormones, in particular the oral contraceptive pill and HRT, in the development of breast cancer is more controversial, but

it can be said with some authority that for most women the benefits of these treatments will far outweigh the small putative risk.

Pathology

Breast cancer may arise from the epithelium of the duct system anywhere from the nipple end of major lactiferous ducts to the terminal duct unit, which is in the breast lobule. The disease may be entirely in situ, an increasingly common phenomenon with the advent of breast cancer screening, or may be invasive cancer. The degree of differentiation of the tumour is usually described by three grades: well differentiated, moderately differentiated or poorly differentiated. Commonly, a numerical grading system based on the scoring of three individual factors (nuclear pleomorphism, tubule formation and mitotic rate) is used, with grade III cancers roughly equating to the poorly differentiated group.

Previously, descriptive terms were used to classify breast cancer (scirrhous, meaning woody, or medullary, meaning brain like). More recently, histological descriptions have been used. These have been shown to have clinical correlations in the way the tumour behaves and are likely to be used for the near future. However, with the increasing application of molecular markers, there will be a change and it is likely that much more information about an individual tumour will be routinely

reported, such as its likelihood of metastasis and to which therapeutic agents it will be susceptible.

Current nomenclature

Ductal carcinoma is the most common variant, but lobular carcinoma occurs in up to 15% of cases. There are subtypes of lobular cancer including the classical type, which carries a better prognosis than the pleomorphic type. Occasionally, the picture may be mixed with both ductal and lobular features. Rarer histological variants, usually carrying a better prognosis, included colloid carcinoma, whose cells produce abundant mucin, medullary carcinoma with solid sheets of large cells often associated with a marked lymphocytic reaction and tubular carcinoma. Invasive lobular carcinoma is commonly multifocal and/or bilateral. Cases detected via the screening programme are often smaller, better differentiated and of special type than those presenting to the symptomatic service.

Inflammatory carcinoma is a fortunately rare, highly aggressive cancer that presents as a painful, swollen breast, which is warm with cutaneous oedema. This is due to blockage of the subdermal lymphatics with carcinoma cells. Inflammatory cancer usually involves at least one-third of the breast and may mimic a breast abscess. A biopsy will confirm the diagnosis and show undifferentiated carcinoma cells. It used to be

rapidly fatal with surgery only hastening the end, but with aggressive chemotherapy and radiotherapy with salvage surgery, the prognosis has improved considerably.

In situ carcinoma is preinvasive cancer that has not breached the epithelial basement membrane. This was previously a rare, usually asymptomatic finding in breast biopsy specimens, but is becoming increasingly common owing to the advent of mammographic screening: it now accounts for over 20% of cancers detected by screening in the UK. In situ carcinoma may be ductal (DCIS) or lobular (LCIS), the latter often multifocal and bilateral. Both are markers for the later development of invasive cancer, which will go on to develop in at least 20% of cases. Although mastectomy is curative, this is over treatment in many cases and the best treatment for in situ carcinoma is the subject of a number of clinical trials. DCIS may be classified by the Van Nuys system, which combines the patient's age, type of DCIS and presence of microcalcification, extent of resection margin and size of disease. Patients with a high score benefit from radiotherapy after excision, whereas those of low grade, who are completely excised, need no further treatment.

Staining for oestrogen and progesterone receptor (ER and PR) is now considered routine, as their presence will indicate the use of adjuvant

hormonal therapy with tamoxifen. Increasingly, tumours are stained for C-erbB2 (a growth factor receptor), as patients can be treated with the monoclonal antibody against this receptor if they relapse.

The pathologist is an important member of the breast cancer team and will increasingly help decide which adjuvant therapies will be appropriate.

Paget's disease of the nipple

Paget's disease of the nipple (Fig.55.27a. and b.) is a superficial manifestation of an underlying breast carcinoma. It presents as an eczema-like condition of the nipple and areola, which persists despite local treatment. The nipple is eroded slowly and eventually disappear. If left, the underlying carcinoma will sooner or later become clinically evident. Nipple eczema should be biopsed if there is any doubt about its cause. Microscopically, Paget's diseases is characterized by the presence of large, ovoid cells with abundant, clear, pale-staining cytoplasm in the Malpighian layer of the epidermis.

THE SPREAD OF BREAST CANCER.

Local spread

The tumour increases in size and invades other portions of the breast. It tends to involve the skin and to penetrate the pectoral muscles and even the chest wall.

Lymphatic metastasis

Lymphatic metastasis occurs primarily to the axillary lymph nodes and to the internal mammary chain of lymph nodes. The site of the tumour within the breast does not dictate which nodes will be involved, for example medial tumours spread just as readily to the axillary nodes as do lateral tumours. The involvement of lymph nodes is not just a chronological event in the evolution of the carcinoma, but rather a marker for the metastatic potential of the tumour. Involvement of supraclavicular nodes and of any contralateral lymph nodes represents advanced disease.

Spread by the bloodstream.

It is by this route that skeletal metastases occur, although the initial spread may be via the lymphatic system. In order of frequency, the lumbar vertebrae, femur, thoracic vertebrae, rib and skull are affected and these deposits are generally osteolytic. Metastases may also commonly occur in the liver, lung and brain and occasionally, the adrenal glands and ovaries, but have been described in most body sites.

Clinical presentation

Although any portion of the breast, including the axillary tail, may be involved, breast cancer is found most frequently in the upper, outer quadrant. Most breast cancers will present as a hard lump, which may be associated with in drawing of the nipple. As the disease advances locally

there may be skin involvement with peau d'orange or frank ulceration and fixation to the chest wall. This is described as cancer-en-cuirasse. About 5% of breast cancers in the UK will present with either locally advanced disease or symptoms of metastatic disease. This figure is nearer 20% in the developing world. These patients must then undergo a staging evaluation so that the full extent of their disease can be ascertained. This will include a careful clinical examination, chest radiograph, serum alkaline phosphatase and gamma-glutamine transaminase (GGT), with liver ultrasound if these are abnormal and an isotope bone scan. This is important for both prognosis and treatment: a patient with widespread visceral metastases may obtain an increased length and quality of survival from systemic hormone or chemotherapy, but she is not likely to benefit from surgery as she will die from her metastases before local disease become becomes a problem. In contrast, patients with relatively small (less than 5cm in diameter) tumours confined to the breast and ipsilateral lymph nodes rarely need staging beyond a good clinical examination as the pick-up rate for distant metastases is so low. Currently, a chest radiograph, full blood count and liver function tests are all that are recommended for screening for patients with early-stage breast cancer.

Peau d’orange

Peau d’orange is due to cutaneous lymphatic oedema, where the infiltrated skin is tethered by the sweat ducts, it cannot swell, leading to an appearance like orange skin. Occasionally the same phenomenon is seen over a chronic abscess.

Late oedema of the arm is a troublesome complication of breast cancer treatment, fortunately seen less often now that radical axillary dissection and radiotherapy are rarely combined. However, it does still occur occasionally after either modality of treatment alone and appears at any time from months to years after treatment. There is usually no precipitating cause but recurrent tumour should be excluded as neoplastic infiltration of the axilla can cause arm swelling due to both lymphatic and venous blockage. This neoplastic infiltration is often painful due to brachial plexus nerve involvement.

An oedematous limb is susceptible to bacterial infections following quite minor trauma and these require vigorous antibiotic treatment. Antibiotics may need to be given for much longer than is normal and patients at risk of infection should have antibiotics readily available in order to start treatment promptly. Treatment of late oedema is difficult but limb elevation, elastic arm stockings and pneumatic compression devices can be useful.

Cancer-en-cuirasse.

The skin of the chest is infiltrated with carcinoma and has been likened to a coat. It may be associated with a grossly swollen arm. This usually occurs in cases with local recurrence after mastectomy, and occasionally is seen to follow the distribution of irradiation to the chest wall. The condition may respond to palliative systemic treatment, but prognosis in terms of survival is poor.

Lymphangiosarcoma

Lymphangiosarcoma is a rare complication of lymphoedema with an onset many years following the original treatment. It takes the form of multiple subcutaneous nodules in the upper limb and must be distinguished from recurrent carcinoma of the breast. The prognosis is poor but some cases respond to cytotoxic therapy or irradiation. Interscapulothoracic forequarter amputation is sometimes indicated.

Staging of breast cancer

There are two traditional systems of classification for breast carcinoma, which predominantly rely on clinical staging of the disease. These are the Manchester system and the International Union Against Cancer, TNM (tumour, nodes, metastases)staging system.

Tx - Primary tumor cannot be assessed

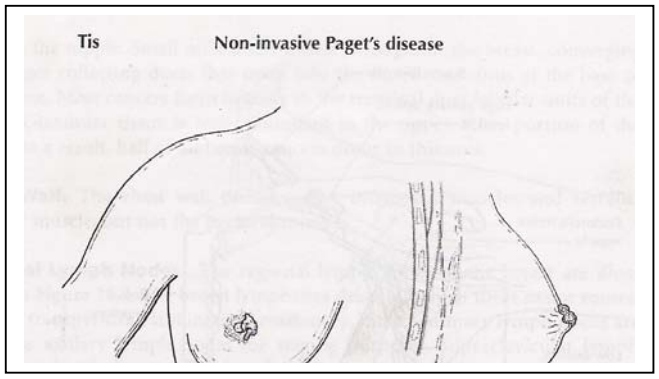
T0 - No evidence of primary tumor

Tis - Carcinoma in situ

Tis - (DCIS) Ductal carcinoma in situ

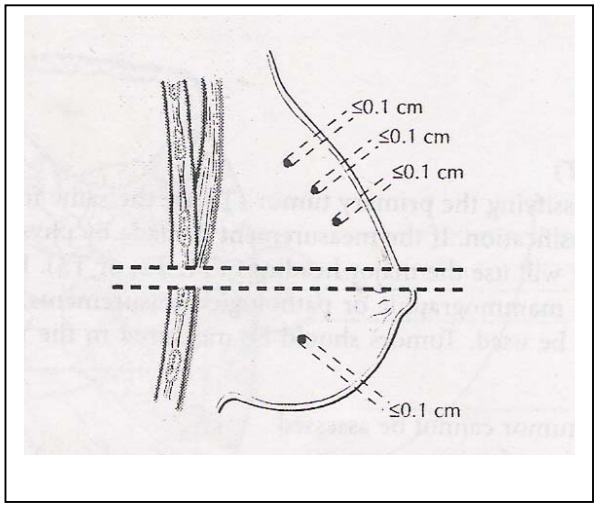
Tis - (LCIS) lobular carcinoma in situ

Tis - (Paget's) Paget's disease of the nipple with no tumour

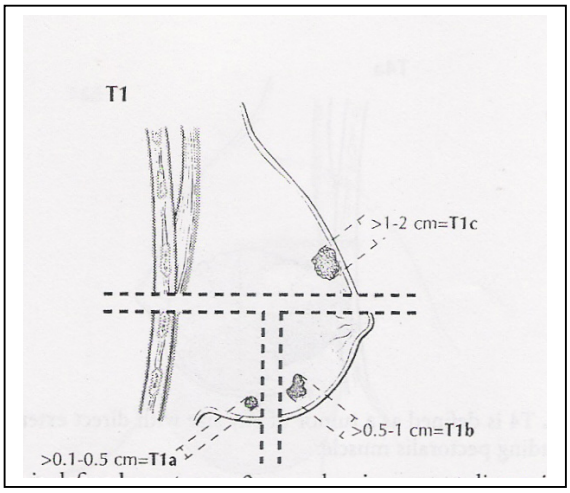


Tis (Paget's) is defined as Paget's disease of the nipple with no tumor

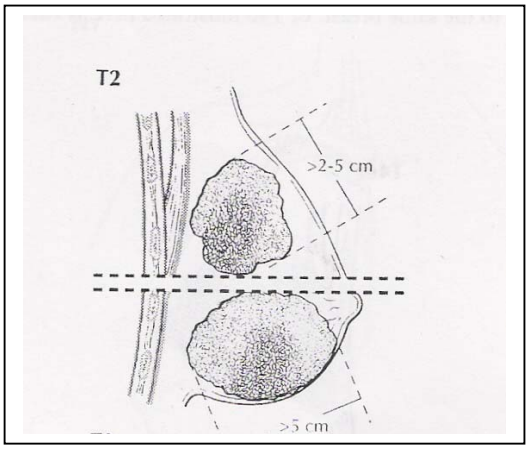
T1 - Tumor 2cm or less in greatest dimension



T1 mic is defined as microinvasion 0.1 cm or less in greatest dimension. The presence of multiple tumor foci of microinvasion should be noted in parentheses



- T1 is defined as a tumor 2 cm or less in greatest dimension.
- T1a is defined as tumor more than 0.1 cm but less than 0.5 cm in greatest dimension :
- T1b is defiend as a tumor more than 0.5 cm but not more than 1 cm in greatest dimension :
- T1c is defined as tumor more than 1 cm but not more than 2cm in greatest dimension.

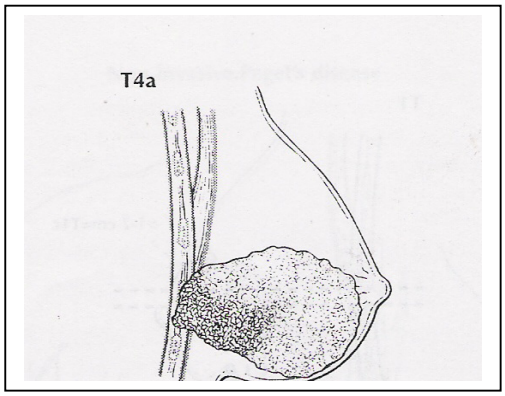


T2 (above dotted line) is defined as tumor more than 2 cm but not more than 5 cm in greatest dimension and T3 (below dotted line) is defiend as tumor more than 5 cm in greatest dimension.

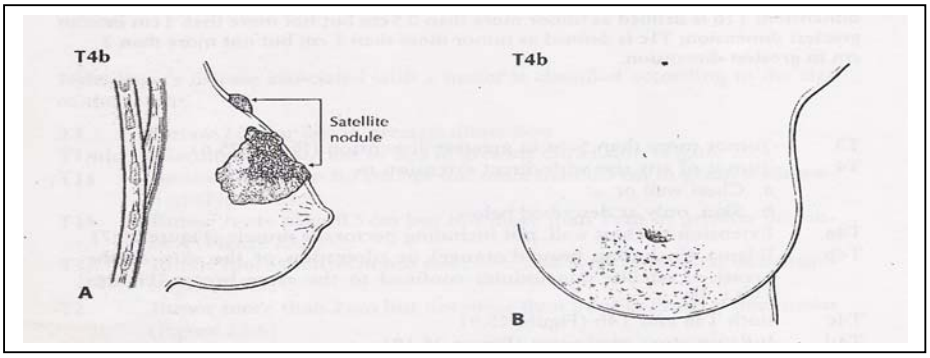
T4 - Tumour of any size with direct extension to

a) Chest wall or

b) Skin, only as described below

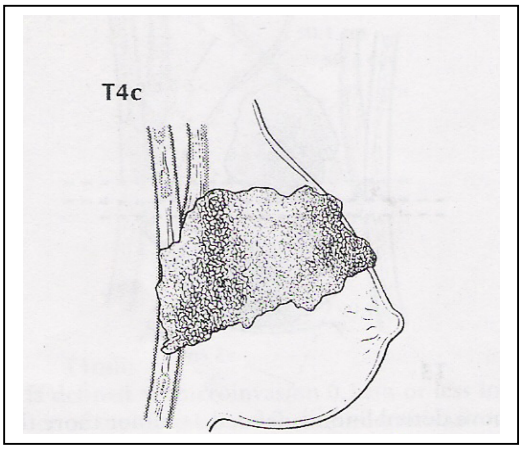


T4a is defined as a tumor of any size with direct extension to chest wall, not including pectoralis muscle

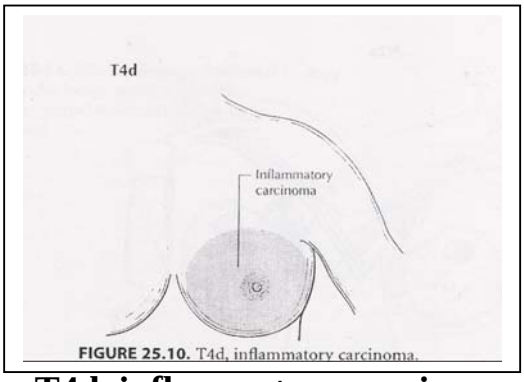


A - T4b, illustrated here as satellite skin nodules, is defined as edema (including peau d' orange) or ulceration of the skin of the breast, or satellite skin nodules confined to the same breast.

B - T4b illustrated here as edema (including peau d'orange)



T4 c is defined as both T4a and T4b

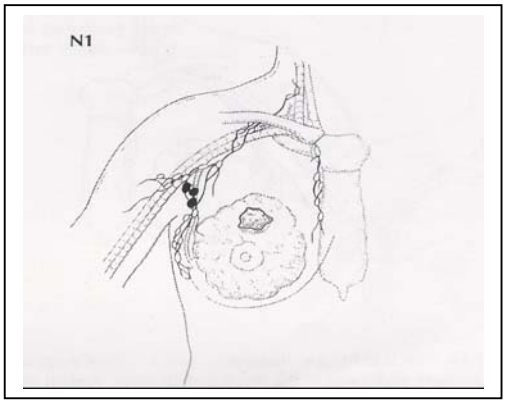


T4d, inflammatory carcinoma

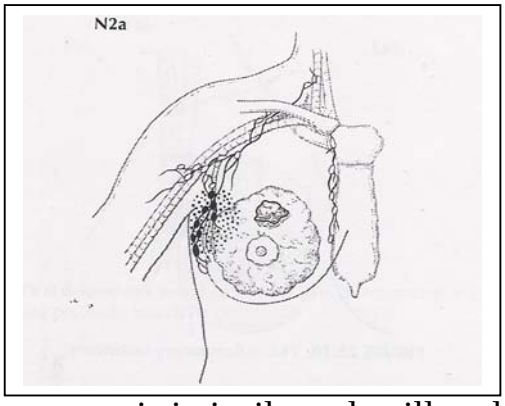
Regional Lymph Node (N) :

Nx - Regional lymph nodes cannot be assessed (eg Previously removed)

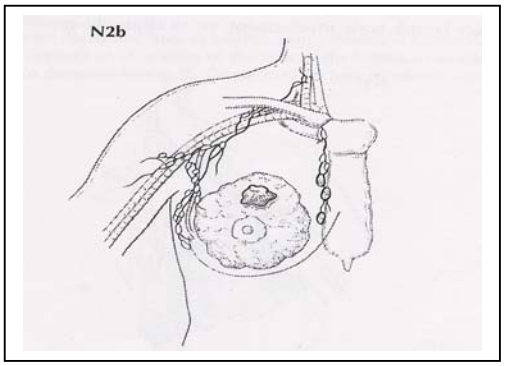
N0 - No regional lymph node metastasis



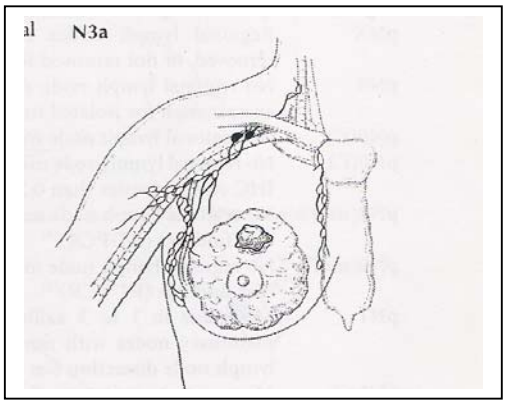
N1 is defined as metastasis in movable ipsilateral axillary node (s)



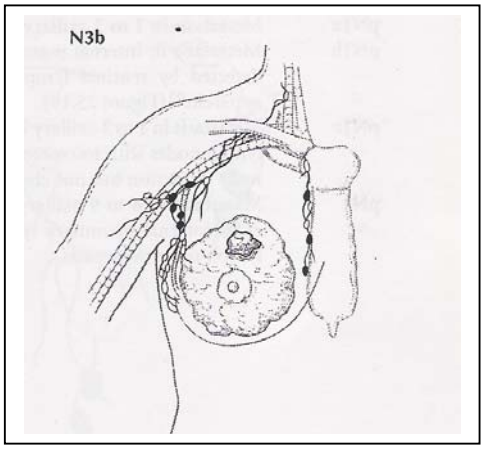
N2a is defined as metastasis in ipsilateral axillary lymph nodes fixed to one another (matted) or to other structures



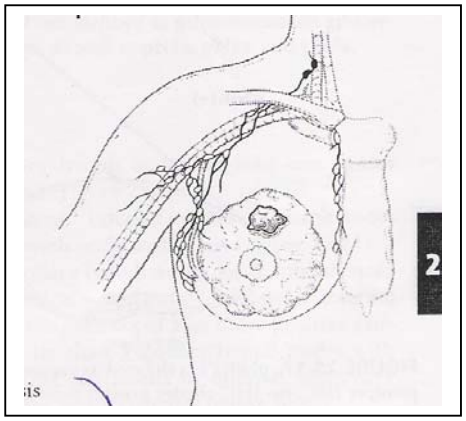
N2b is defined as metastasis only in clinically apparent ipsilateral internal mammary nodes and in the absence of clinically evident axillary lymph node metastasis.



N3a metastasis in ipsilateral infraclavicular lymph nodes without axillary or internal mammary lymph node involvement



N3b metastasis in ipsilateral internal mammary lymphnode and axillary lymph nodes



N3c is defined as metastasis in ipsilateral supraclavicular lymph nodes

Mx - Distant metastases cannot be assessed

M0 - No evidence of distant metastases

M1 - Presence of distant metastases

STAGE GROUPING

	T0	T1	T2	T3	T4
No		I			
N1		IIa	IIb		IIIb
N2		IIIa			
N3			IIIc		
M1		IV			

Prognosis of breast cancer

The best indicators of likely prognosis in breast cancer are still tumour size and lymph node status. However, it is realised that some large tumours will remain confined to the breast for decades, whereas some very small tumours are incurable at diagnosis. Hence the prognosis of a cancer depends not on its chronological age but on its invasive and metastatic potential. Although prognosis broadly correlates with stage, other factors also influence prognosis and should be assessed, for example the Nottingham Prognostic Index includes not only tumour size and lymph node status but also tumour grade. This has been validated in many centres and consists of a score given by the formulae I= (0.2x size)

+ grade + nodes. The size is in centimeters, the grade is on a 1-3 score and the nodes are also scored on 1-3 where a score of one indicates no nodal involvement, two indicates on the there nodes involved. Based on the overall index, patients can be divided into an excellent prognosis group, a moderate prognosis group and a poor prognosis group. The chance of dying from breast cancer in the first group is so low that many patients do not require additional treatment.

Equally, the use of adjuvant systemic therapy is decided not only on tumour size and lymph node status but also biological measures such as oestrogen receptor status, patient age and menopausa status. Tamoxifen can be recommended irrespective of clinicopathological variable if the patient is hormone receptor positive.

Thus, as we gain more knowledge of the biological variables that affect prognosis, it becomes increasingly clear that it is these factors ***(***Histological grade of the tumour, hormone receptor status, measures of tumour proliferation such as S-phase fraction and thymidine labeling index, growth factor analysis and oncogene or oncogene product measurements***.),*** rather than anatomical mapping, which influence outcome and treatment. Perhaps a more pragmatic approach would be to classify patients according to the treatment that they require.

Pragmatic classification for breast cancer

Group	Approximate 5–year survival	Example	Treatment
‘very-low-risk primary breast cancer	>90%	Screen-detected DCIS, tubular or special types	Local
‘Low-risk primary breast cancer	70-90%	Node negative with favourable histology	Locoregional with/without systemic
‘High-risk primary breast cancer	<70%	Node positive with unfavourable histology	Locoregional with systemic
Locally advanced	<30%	Large primary or inflammatory	Primary systemic
Metastatic	-	-	Primary systemic

Early Breast cancer : Stage I, IIA & IIB

All patients should be treated using a protocol based multi – modality treatment plan. The primary modality for treating EBC is surgery augmented by adjuvant radiation therapy to improve loco regional control of the disease and systemic therapy in the form of chemotherapy and hormone therapy for improving systemic control and overall survival of the patients.

The two components of primary surgical treatment of the EBC – Management of the breast primary and the axilla – almost always go hand in hand. With better understanding of the disease process recognition of fact that the survival of breast cancer patients does not depend on the

radicality of the breast surgical procedure and use of evidence based medicine tools to test appropriateness of lesser radical surgical procedure in management of EBC, the world has seen a dramatic and progressive conservation in management of the breast primary as well as the axilla.

While standard surgical procedure for breast primary has changed from a radical mastectomy to a wide local excision in appropriate patients over past 4 decades or so, the management of axilla has seen a paradigm shift from a routine complex axillary dissection in all patients to avoidance of axillary dissection by use of sentinel lymph node biopsy (SLNB) in selected patients with clinically impalpable axillary lymphnode in whom the SLNB is reported non metastatic on histology.

Breast Surgery options for EBC : Whole Breast removal options

1. Modified radical mastectomy with a routine axillary clearance
2. Total mastectomy and SLNB, proceed to axillary clearance if sentinel lymph node is reported metastatic on histology.

BREAST CONSERVATION OPTIONS :

1. Wide local excision with 1 cm clearance all around and occasionally quadrantectomy with routine axillary clearance.
2. WLE or quadrantectomy with SLNB, proceed to axillary clearance if sentinel lymph node is reported metastatic on histology.

INDICATIONS FOR BCT :

1. Choice of the patient
2. Size of the tumor < 4 cm / mammographically detected lesions
3. Clinically negative axillary nodes or N1
4. Well differentiated tumors

Contraindications for BCT :

Absolute :

1. Persistent positive margin > 2 times
2. Previous RT (poor skin tolerance)
3. Multicentricity
4. Pregnancy

Relative :

1. Collagen vascular disease
2. Tumor breast ratio
3. Poorly differentiated tumors

Radiotherapy in BCT :

A post operative irradiation to the breast with / without boost to the tumor bed is a must following breast conservation surgery. Radiotherapy may be

1. **Whole breast irradiation** with a dose of 45-50 Gy given as 2 Gy/day, 5 days a week over 5 weeks & boost to region of tumor bed.

Radiation boost to tumor bed can be delivered either with interstitial implants or using electron or photon beam external radiation.

2. Accelearated partial breast Irradiation :

Post lumpectomy RT exerts its maximal effect on eradicating residual disease in the region of tumour bed and that areas of occult disease in the remainder of the breast is of little practical significance.

This hypothesis provides basis for the use of accelerated partial breast irradiation which is a NEW treatment option. In this technique, larger daily doses of RT is administered only to portion of breast containing primary tumor over 4-5 days, in contrast to 5 week course required for whole breast irradiation, by Brachytherapy (Interstitial, intracavitary), limited EBRT, intra operative limited RT.

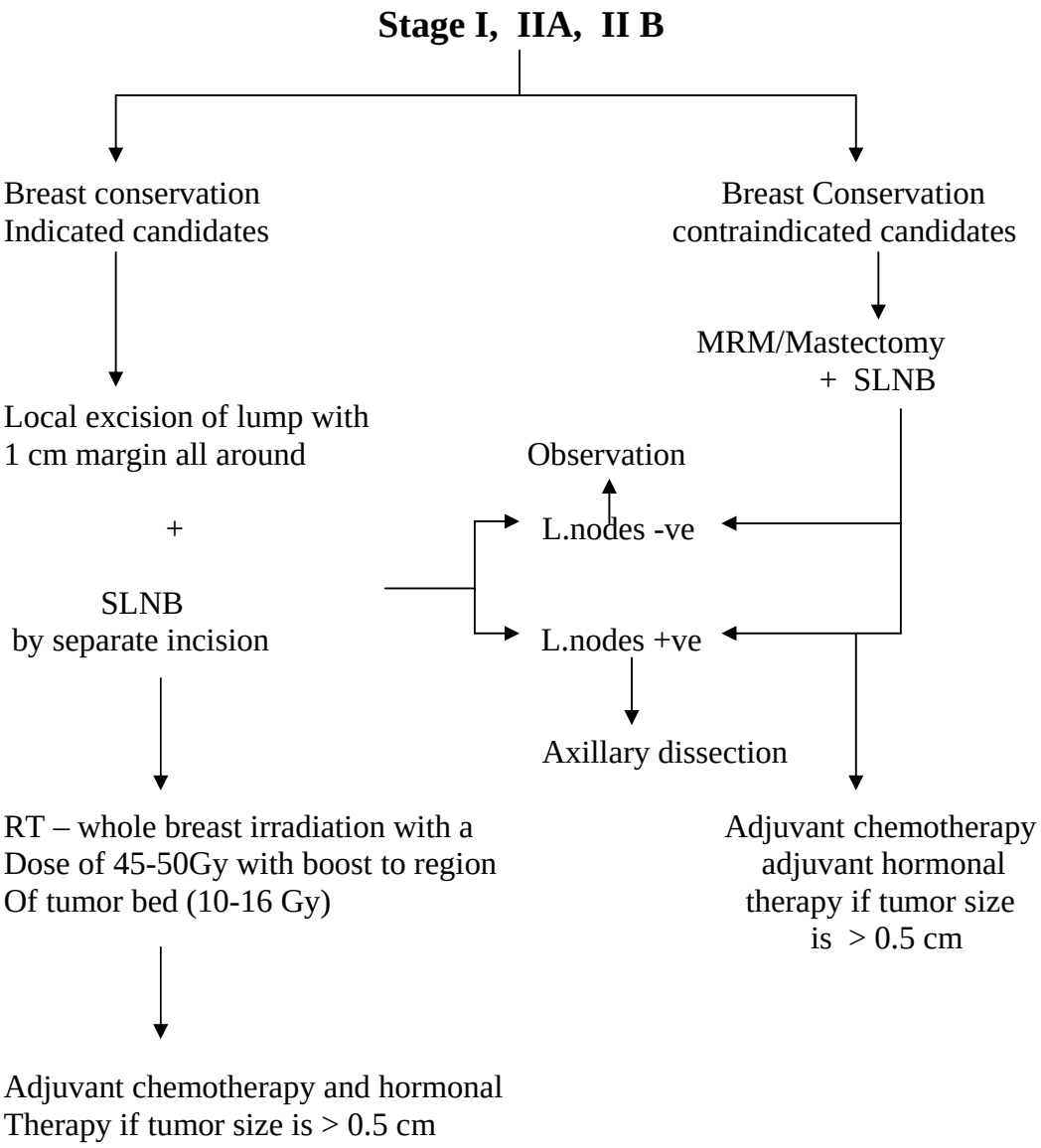
Recurrence after BCT :

The local recurrence rates after lumpectomy and radiation therapy are now less than 5% at 10 years in many large centres. The types of recurrences are

1. True Recurrence : Recurrences at or near primary site (with in boosted region)
2. Marginal Miss : Adjacent to boosted region
3. Elsewhere : Occuring at a distance from original tumor and prerumably representing a new primary.

The choice between a mastectomy procedure and BCT rate on variable factors and all patients desirous of and who do not have any contraindications for BCT and radiation therapy should be offered Breast conservation surgery. Yet mastectomy continues to be the commonest form of curative surgical procedure due to various reasons including low acceptability on patient's part and lack of adequate radiotherapy and pathology infrastructure, training and skills at most centres in India and other developing countries.

Management Protocol for early breast cancer



LOCALLY ADVANCED BREAST CANCER

The definition of LABC has evolved from that of Haagensen and Stout, to encompass a wide spectrum of clinical presentations :

- Large tumors (> 5 cm)
- Extensive regional lymph node involvement
- Direct involvement of the underlying chest wall or skin with edema (including peau d'orange) or ulceration or satellite skin nodules confined to the same breast. Other discrete skin changes, such as dimpling or nipple retraction, may occur in T1-3 disease; they do not constitute evidence of a locally advanced tumor.
- Tumors considered inoperable but without distant metastasis (including involvement of the supraclavicular lymph nodes)
- Inflammatory breast cancer (IBC)

All T and N permutations included in stage II B, III or IV comprised many distinct substage possibilities. The presence of T4 or N3 or regional M1 lesions would result in inclusion in the stage IIIB / IV unresectable subcategory. Most of the patients with either T3 or N2 but without T4, N3 or regional M1 lesions, are included in the stage II / IIIA or operable subcategory. LABC also includes T2 tumors that are too large in proportion to the size of the breast. The clinical diagnosis of LABC is usually not difficult. Patients uniformly present with a large

breast mass. Other symptoms often reported are edema, redness, nipple retraction, pain, skin dimpling, an axillary mass and breast ulceration. Most physical findings are obvious upon inspection or palpation. However, in younger women, some tumors infiltrate the breast diffusely and a discrete mass is difficult to palpate. More than 75% of patients have clinically palpable axillary and / or supraclavicular adenopathy, and 65% - 90% of patients have pathologically confirmed lymphnode metastasis ; > 50% have more than four nodes involved. Most of the LABCs are operable ; only 25%-30% are diagnosed at an inoperable stage.

A core needle biopsy is quite effective in establishing the diagnosis and also allowing tumor samples to be obtained for hormone receptors, DNA studies and other biomarkers.

Appropriate staging procedures should be performed in patients with LABC since the probability of distant metastases is high. Approximately 20% of these patients, appropriately staged, have detectable distant metastases at the time of diagnosis.

After the physical examination and bilateral mammogram, the following additional tests are recommended ; a biochemical profile, including tests of liver and renal function, and calcium level ; chest x ray; bone scans ; radio graphs of areas that appear to be abnormal on the bone

scan ; computed tomography of the liver and an ultrasonography of the breast and regional lymph nodes to precisely assess the tumor extent. The importance of an accurate initial assessment of the extent of primary tumor burden cannot be overemphasized since the efficacy of subsequent local treatment will depend mostly on this initial assessment.

1. Patients with LABC are more prone to develop occult micrometastases elsewhere at the time of presentation.
2. In view of extensive skin involvement, local recurrence is common and adequate skin clearance is not possible.

Hence all patients with LABC are immediately exposed to systemic therapy (Neo adjuvant).

Principles of Neo-adjuvant Treatment :

1. Decreases the incidence of local recurrence
2. Controls the micrometastases at the outset.
3. Helps to assess the response of individual patients to systemic chemotherapy in vivo which is a strong prognostic factor.
4. Causes shrinkage of tumor thereby facilitating skin closure.

Following Neo-adjuvant treatment patients are assessed using RECIST criteria.

(Response Evaluation Criteria In Solid Tumors) based on clinical & radiological examination and the response is classified as

CR - Complete resolution clinically and radiologically which persist for more than 4 weeks

PR - > 50% response clinically and image wise

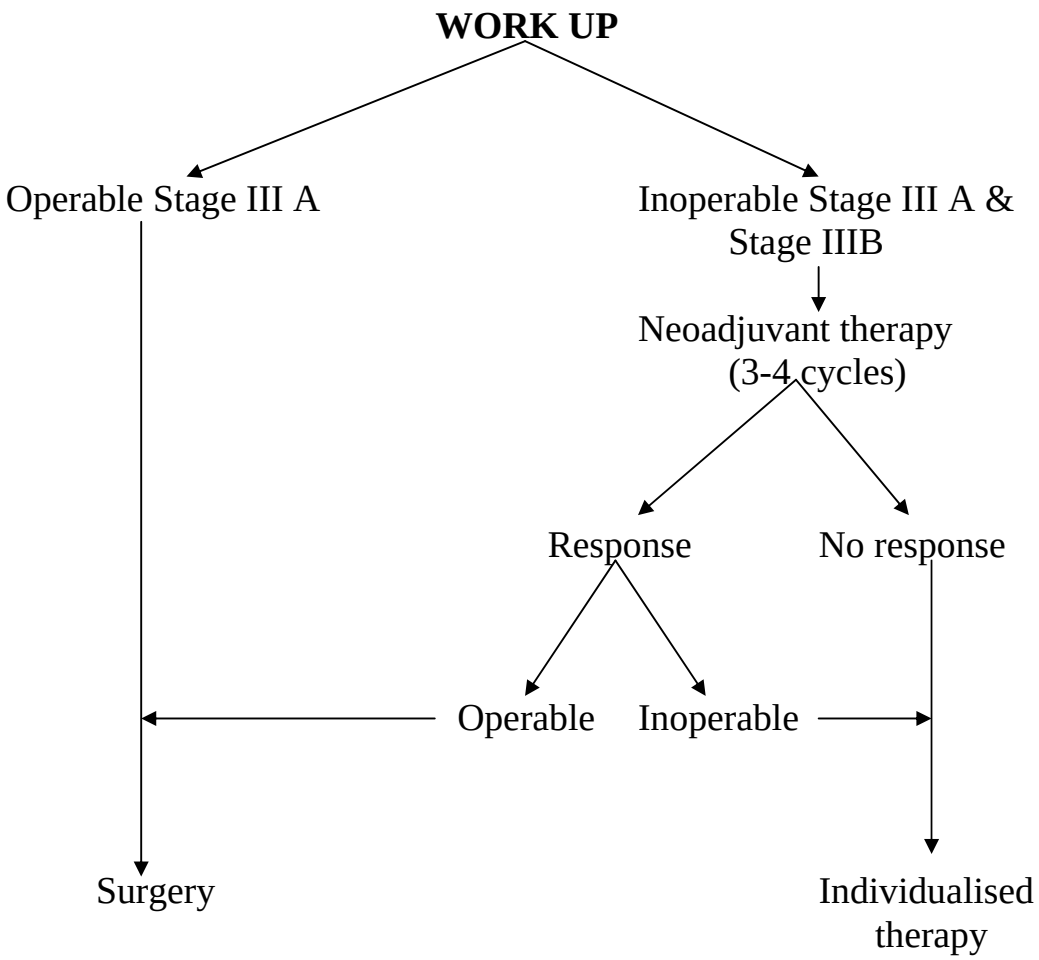
NR - < 50% response

Because physical examination and mammography do not adequately predict the pathologic response to neoadjuvant chemotherapy, alternative imaging methods have been developed to attempt to more accurately predict a pathologic response and to improve breast conservation rates. MRI is one promising modality and appears to correlate well with pathologic response.

LABC

TREATMENT PATHWAYS

Clinically stage III A, IIIB & IIIC Breast Cancer



Operable LABC :

Patients with stage III A disease should undergo modified radical mastectomy (MRM), and adjuvant chemotherapy and locoregional radiotherapy if feasible. They may be managed with MRM followed by chemotherapy and locoregional radiotherapy, or chemotherapy first followed by MRM and locoregional radiotherapy. Breast conserving surgery is currently not a standard approach.

In the trails that compared preoperative chemotherapy with chemotherapy administered post operatively, the proportion of women with tumours greater than 5 cm in diameter ranged from 5% to 27%. Patients with operable stage III disease who desire to preserve their breast should be made aware that BCS is currently not a standard approach and is generally not recommended.

Choice of chemotherapy :

Chemotherapy should contain an anthracycline. Acceptable regimens are 6 cycles of FAC, CAF, CEF or FEC.

Inoperable LABC :

Patients with stage IIIB or IIIC disease, including those with inflammatory breast cancer and those with isolated ipsilateral internal

mammary or supraclavicular lymph-node involvement, should be treated with primary anthracycline – based chemotherapy.

Acceptable chemotherapy regimens are FAC, CAF, CEF or FEC.

CHEMOTHERAPY COMBINATIONS

FAC chemotherapy :

5 – Fluorouracil 500 mg / m^2 IV days 1 & 8 or days 1 & 4

Doxorubicin 50 mg/m^2 IV day 1 (or by 72 hrs continuous infusion)

Cyclophosphamide 500 mg/m^2 IV day 1 cycled every 21 days for 6 cycles

CAF chemotherapy :

Cyclophosphamide 100 mg/m^2 PO days 1-14

Doxorubicin 30 mg/m^2 IV days 1 & 8

5-Fluorouracil 500 mg/m^2 IV days 1 & 8 cycled every 28 days for 6 cycles

AC chemotherapy :

Doxorubicin 60 mg /m^2 IV day 1

Cyclophosphamide 600 mg/ m^2 IV day 1

Cycled every 21 days for 4 cycles

FEC chemotherapy :

Cyclophosphamide 75mg/ m^2 PO days 1-14

Epirubicin 60 mg / m^2 IV days 1 & 8

5 – Fluorouracil 500 mg / m^2 IV days 1 & 8

With cotrimoxazole support. Cycled every 28 days for 6 cycles

Preoperative radiation therapy was often able to convert an inoperable breast cancer to an operable one. Preoperative radiation therapy did not seem to differ from post operative radiation in providing additional locoregional control. A combination of surgery and radiation therapy provided the maximum chance for locoregional control over high dose radiation therapy or surgery alone.

When locoregional radiotherapy is delivered following MRM for locally advanced disease, radiation should be delivered to the chest wall, supraclavicular and axillary nodes. Whether treatment to the internal mammary nodes is required is unclear. The recommended dose of radiation is 50 Gy in 25 fractions or equivalent.

- Patients with stage III B disease who respond to chemotherapy should receive surgery plus locoregional radiotherapy
- The locoregional management of patients with stage III C disease who respond to chemotherapy is unclear and should be individualized.

- Patients whose disease remains inoperable following chemotherapy should receive locoregional radiotherapy and subsequent surgery if feasible.

Patients who are treated primarily with radiotherapy should be given tumouricidal doses to areas of bulk disease (60-66Gy in 30 to 33 fractions or equivalent) Higher doses of radiation (70 Gy in 35 fractions by external beam or brachytherapy) to areas of bulk disease may be considered for patients if surgery is felt not to be an option and if tolerance of critical organs permits. Two case series have reported a doseresponse relation with higher doses of radiation that resulted in decreased rates of local recurrence.

For the patient who has a partial or complete response to chemotherapy and whose lesion is converted to an operable state, the next manoeuvre is typically mastectomy to debulk gross disease, to facilitate local-regional control, and to allow for the pathologic assessment of response. For patients with a complete or partial response, the optimal chemotherapy to use after local-regional treatment is uncertain. Specifically, it is not clear whether to continue the same chemotherapy as before after local-regional treatment or whether a cross-resistant chemotherapeutic regimen is indicated. The ASCO guidelines

recommend postmastectomy radiation treatment, in general, for those patients who require a mastectomy

For the patient whose tumor remains inoperable after first-line systemic chemotherapy, the options are to proceed with second-line chemotherapy or to deliver preoperative radiation treatment. One major goal of treatment is to attempt to convert the lesion from an inoperable to an operable state, because patients without local – regional control have substantially diminished quality of life.

Inflammatory Breast Cancer :

Inflammatory breast cancer is a distinct clinical subtype of locally advanced breast cancer, with a particularly aggressive behavior and poor prognosis. Clinically, inflammatory breast cancer typically presents with the rapid onset of breast erythema, warmth, and edema, often without a discrete underlying mass. The swelling of the breast can be quite pronounced, producing significant tenderness. Although histologic proof of malignancy is critical prior to treatment of IBC, documenting dermal lymphatic permeation is not critical in establishing the diagnosis of IBC.

Bonnier et al classified patients into three groups according to clinical and histopathological features.

Group A included patients with typical inflammatory breast cancer (diffuse enlargement of the breast, often no palpable tumour, redness and

oedema of the skin) Ipsilateral enlargement of the axillary nodes was often detected and emboli of carcinoma cells in the subdermal lymphatics were often found.

Group B included patients with occult inflammatory breast cancer, in which the presence of tumour emboli in dermal lymphatics was not associated with inflammatory symptoms and signs.

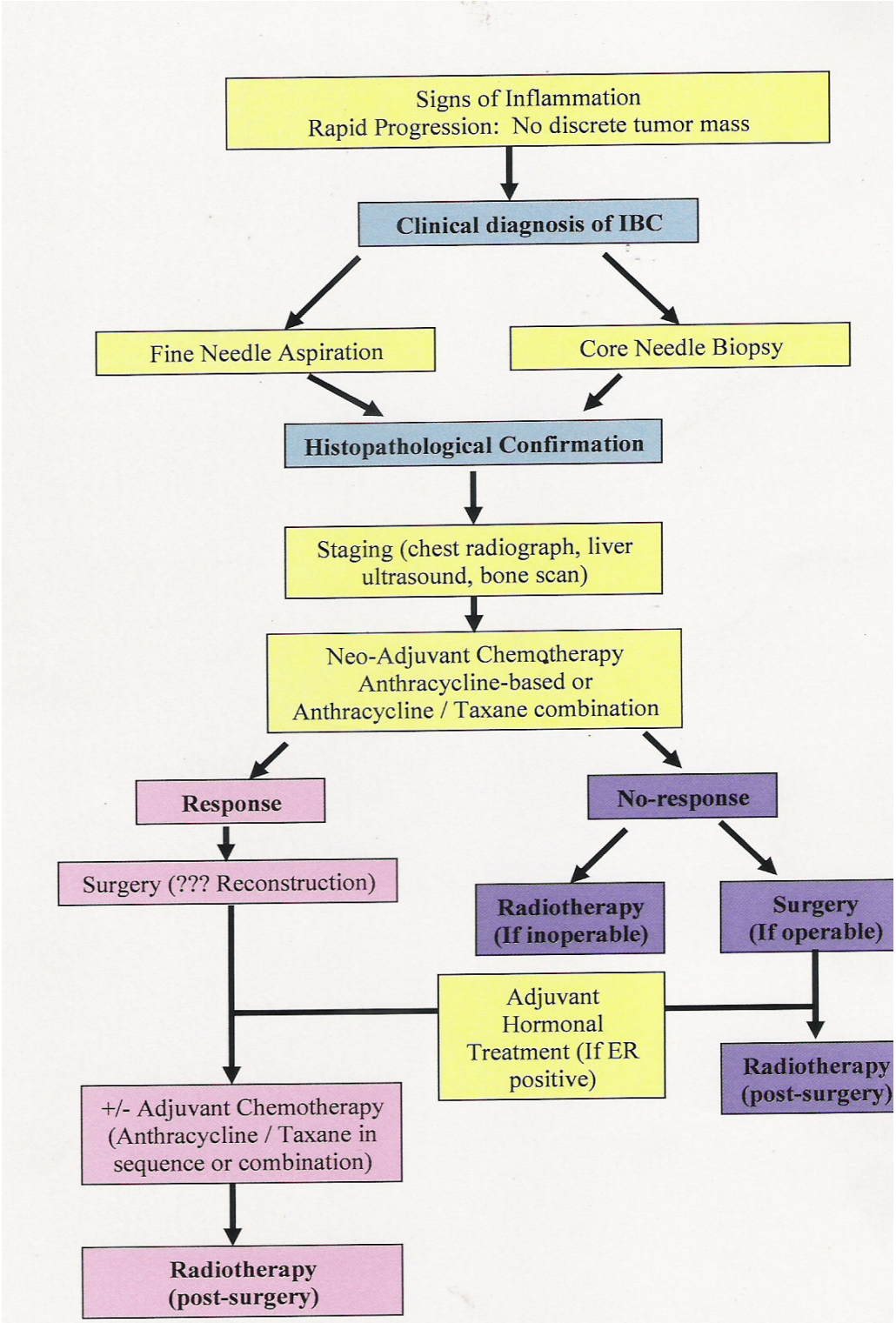
Group C included patients with pseudo-inflammatory breast cancer. Symptoms were similar to those of groupA. However a tumour mass was more readily palpable and the sub-dermal lymphatics were never involved Furthermore, the axillary nodes were rarely involved.

Neglected locally advanced breast cancer can develop secondary inflammatory characteristics, but should be distinguished from primary inflammatory carcinoma as these secondary inflammatory breast cancers may follow a more indolent course and can be treated as other locally advanced breast carcinomas. Three biological features make inflammatory breast cancer a unique clinical entity:

(1) Rapidity of progression

(2) High angiogenic and angioinvasive capability

(3) Aggressive behavior from inception.



Algorithm for the diagnosis and treatment of "inflammatory" breast cancer

Advanced Breast Cancer : Stage IV

Metastatic spread of breast cancer is invariably associated with death from the disease. The median survival from the manifestation of metastases is about 3 years in most reported series. Metastatic disease can be discovered is less than 5% to 10% of the newly diagnosed cases of breast cancer. In most cases, metastasis can be detected during surveillance after the course of initial therapy for the primary cancer. About 75% of patients who develop metastatic disease after initial treatment of primary will do so within 5 years of diagnosis of the primary tumor. While managing the metastatic breast cancer, all efforts should be made to provide adequate palliation and to extend the survival wherever possible.

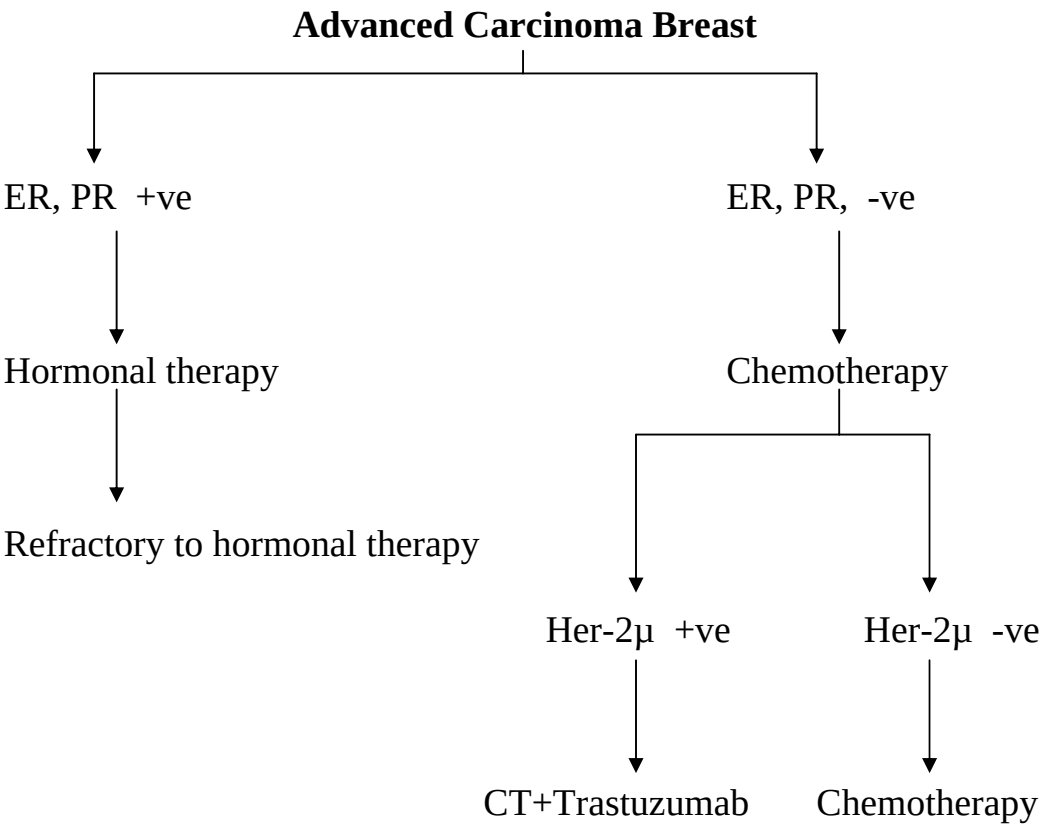
The most common sites of distant mets are bone (49% to 60%), lung (15-20%), pleura (10%-18%). Soft tissues (7% to 15%) and liver (5% - 15%). Routine history and careful physical examination can detect 91.8% of all recurrences.

Prognostic factors in Advanced Breast Cancer :

1. Tumor biology (Grade, ER, PR status, Her 2 µ status)
2. Performance status
3. Cancer related symptoms
4. Site of recurrence
5. Number of sites of recurrence
6. Prior adjuvant therapy
7. Disease free interval
8. Prior therapy for metastatic disease
9. Response / duration of treatment with prior therapy for metastatic disease.

No.	Good Prognosis	Bad prognosis
1.	Patient who received less therapy	Heavily treated patients with
2.	Long disease free interval since diagnosis	shorter interval since treatment
3.	Soft tissue / bone mets	Visceral mets
4.	Fewer symptoms	Greater symptomatology
5.	Better performance status	-
6.	ER, PR positive	-

MANAGEMENT PROTOCOL



Surveillance following initial management of breast cancer :

American Society of clinical Oncology Guidelines: Survelliance for Breast Cancer Follow-up[o]

Test	**Frequency**
History, eliciting of symptoms,	Every 3-6mg X 3y,
Physical examination	Every 6-12 mo X2y, then annually
Breast self examination	Monthly
Mammography (contralateral & Ipsilateral)	Annually
Pelvic examination	Annually
Complete blood counts, liver chemistry,	Not recommended
Serum Tumour markers (CA 19-3, CEA)	Not recommended
X-ray chest, Bone scan	Not recommended
USG of liver, CT scan of chest, Abdomen and pelvis	Not recommended

Hormonal therapy :

The various modalities of endocrine manipulation available in the management of advanced breast cancer include.

Selective Estrogen Receptor Modulators:

1.Tamoxifen

2.Torimefen

Androgens : Fluoxymesterone

Progrestins : Megestrol acetate

Medroxyprogesterone aceate

High dose Estrogens

Aromatase inhibitors: 1st generation: Aminoglutethemide

2nd generation : Formestane (Type 1), Fadrzole

3rd generation: Exemestane (Type1), Anastrazole,

Letrozole, Vorozole

Steroidal Antiestrogens: Fulvestrant

LHRH agonists: Leuprolide, Goserelin

Gland ablation:

Surgical (open/laparoscopic): chemical; radiation

Ovary; Pituitary; Adrenals

Targeted therapy – recent advances :

Advances in molecular biology are reaching therapeutic application on several fronts. One example is the targeting of the HER-2 tyrosine kinase receptor. Trastuzumb (Herceptin, Genentech, San Francisco) is a humanized monoclonal antibody that binds to HER-2 with great affinity, resulting in growth arrest of HER-2 over expressing cancer cells. The addition of trastuzumab to AC and paclitaxel improves time to progression, response rates, and overall survival for patients with advanced breast cancer over expressing HER-2. Monoclonal antibodies are large molecules and are likely to be more effective in the adjuvant setting. Randomized trials that integrate trastuzumab in combination with chemotherapy are under way.

MATERIALS AND METHODS

277 cases of carcinoma breast admitted in Govt. Rajaji Hospital, Madurai Medical College, Madurai during the period of December 2007 to November 2009 were studied out of which 4 were male patients and they were excluded from the study.

A detailed history has been taken and thorough general examination was made and cases were studied as per the proforma attached.

These patients were analysed according to age, clinical Presentation, stage of the disease, menopausal status.

A special attention was given to LABC since it formed the major bulk of my study group where neo adjuvant therapy was given.

RESULTS

Age Distribution

Age in Years	Cases	
	No.	%
Up to 30	18	6.6
31-40	72	26.4
41-50	86	31.5
51-60	65	23.8
61-70	27	9.9
> 70	5	1.8
Total	273	100

In this group, maximum number of patients were found in the age group of 41-50 years whereas the incidence was less in the extremes of age.

Stage at the time of Presentation

Type of Breast Cancer	Cases	
	No.	%
Early breast cancer I & II	119	43.6
Locally advanced breast cancer III (O) + III A IO -) + III A (IO) + III B (Down staged) + III B (IO)	118	43.2
Advanced Carcinoma breast IV	36	13.2
Total	273	100

In this study group, 43.6% belong to early breast cancer and an equivalent percentage to locally advanced breast cancer. 13.2% of patients were in the advanced stage.

Menopausal Status

Menopausal Status	Cases	
	No.	%
Pre menopausal	144	52.9
Post menopausal	129	47.3
Total	273	100

In this study, 52.9% were pre menopausal and 47.3% were post menopausal.

Age wise distribution of Staging

Age group	Staging								
	I	II	III A (o)	III A (IO) O	III A IO	III B	III B IO	IV	Total
Up to 30	7	1	3	3	0	0	2	2	18
31-40	14	20	10	8	0	6	5	9	72
41-50	2	41	24	4	1	3	3	8	86
51-60	2	22	20	9	1	0	3	8	65
61-70	1	9	8	2	0	0	1	6	27
> 70	0	0	2	0	0	0	0	3	5
Total	26	93	67	26	2	9	14	36	273

Incidence of operability

Staging	Cases	
	No.	%
Operable Carcinoma		
I	26	9.5
II	93	34.1
III A (O)	67	24.5
III A (IO) – (O)	26	9.5
III B	9	3.3
Total Operable	221	81
Inoperable		
III A (IO)	2	0.7
III B (IO)	14	5.1
IV	36	13.2
Total inoperable	52	19
Total	273	100

In this study group, 81% of the patients were found to be operable. This includes patients with LABC who are initially inoperable but downstaged and became operable. 19% of the patients were inoperable.

Comparison between our study and SEER study of National Cancer Institute, US. (2002 – 2006)

Stage of Breast Cancer	Our study	SEER Study
I	9.5%	60%
II & III	77.2%	33%
IV	13.2%	5%

Age at Diagnosis	Our study	SEER Study
Under 20 years	0 %	0 %
20 – 34 yrs	11%	1.9%
35 – 44 yrs	28.2%	10.5%
45-54 yrs	28.9%	22.5%
55 – 64 yrs	22.3%	23.7%
> 65 yrs	9.5%	41.3%

DISCUSSION

273 cases of Carcinoma breast, admitted in Government Rajaji Hospital, Madurai are presented in this dissertation. The maximum age incidence of Carcinoma breast in the study group has been 41-50 years of age.

In this study group, about 9.5% of the patients were with stage I disease. This when compared to the SEER study of National Cancer Institute,US was very low where stage I accounted for 60%. This reflects the need for spreading the awareness among women regarding self breast examination and utilizing screening programme.

Locally advanced breast cancer accounted for 77.2% where as it is only 33% in SEER study which again reinforces the need for improvement in health education. Stage IV disease accounted for 13.2% and 5% in my study and SEER study respectively.

About 68.2% of patients in the study group were operable initially at the time of admission itself.

18.6% of patients were inoperable at the time of admission, out of which 12.8% of patients were downstaged and became operable and 5.9% of patients remained inoperable even after downstaging.

Down staging was done with 3 cycles of CAF regimen and in some cases with chemo RT and response was assessed both clinically and radiogically with mammogram.

All patients were assessed preoperatively by clinical examination, biopsy and for metastatic work up chest x-ray and USG abdomen were done.

All operable patients underwent modified radical mastectomy and followed by adjuvant therapy with either chemotherapy or radiotherapy or both and hormonal therapy.

About 81.2% of patients were operable including those patients who were downstaged.

To conclude one of the important factors of fight against breast cancer is breast cancer awareness. The more knowledge known about the disease and the more done to check for early warning signs, the better chance for survival. Research and development continues to be done so that one day we can say the history of breast cancer is over.

CONCLUSION

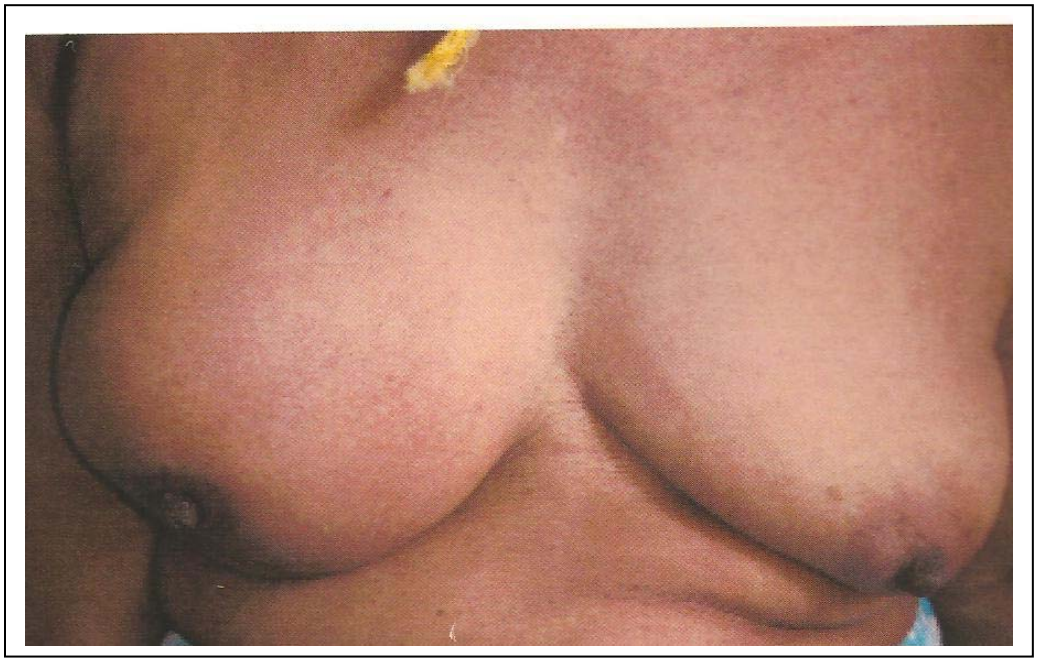
The incidence of stage I breast cancer in this study group is only 9.5% whereas it is 60% in the SEER study of National Cancer Institute (NCI) of United States. LABC accounted for 77.2% in the study group whereas its only 33% in SEER study. The significance of this conclusion is that what cases are classified at a specific instance as LABC once belonged to the category of early breast cancer and subsequently evolved into LABC due to either patient's lack of awareness about the disease or inappropriate interventions or aggressive tumor biology. Thus as prevention is always better than cure it is recommended that the follow up measures can be adopted to address this problem.

1. Improving Health education regarding self breast examination
2. Screening mammography
3. General education about early symptoms of the disease and access to medical facilities are important in diminishing breast cancer incidence.
4. Identification of high risk population and specific management
5. Surveillance when family history is positive for breast cancer. Metastatic work up is mandatory.

T 4 TUMOUR – SHINY SKIN +

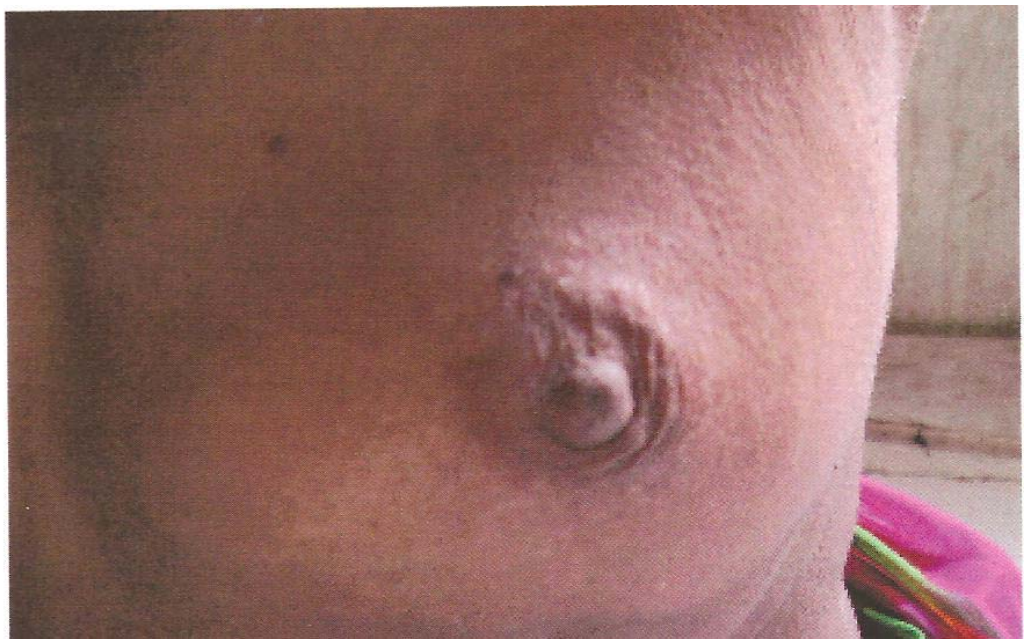
FUNGATING CARCINOMA (T4C)

T4B TUMOUR

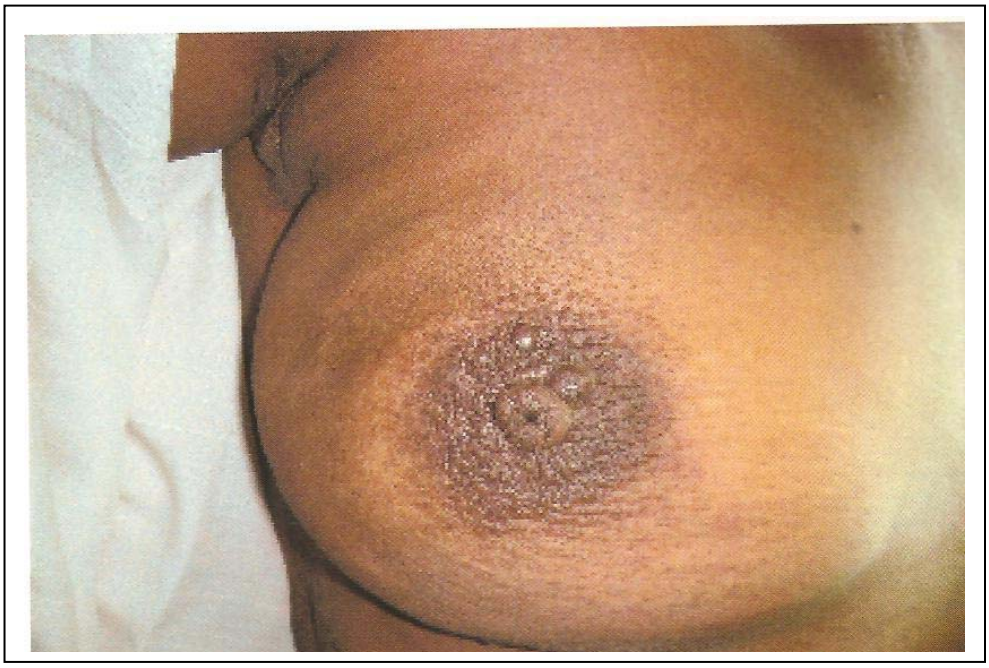


NIPPLE RETRACTION

T3 TUMOUR



SATELLITE NODULES +



PEAU D' ORANGE

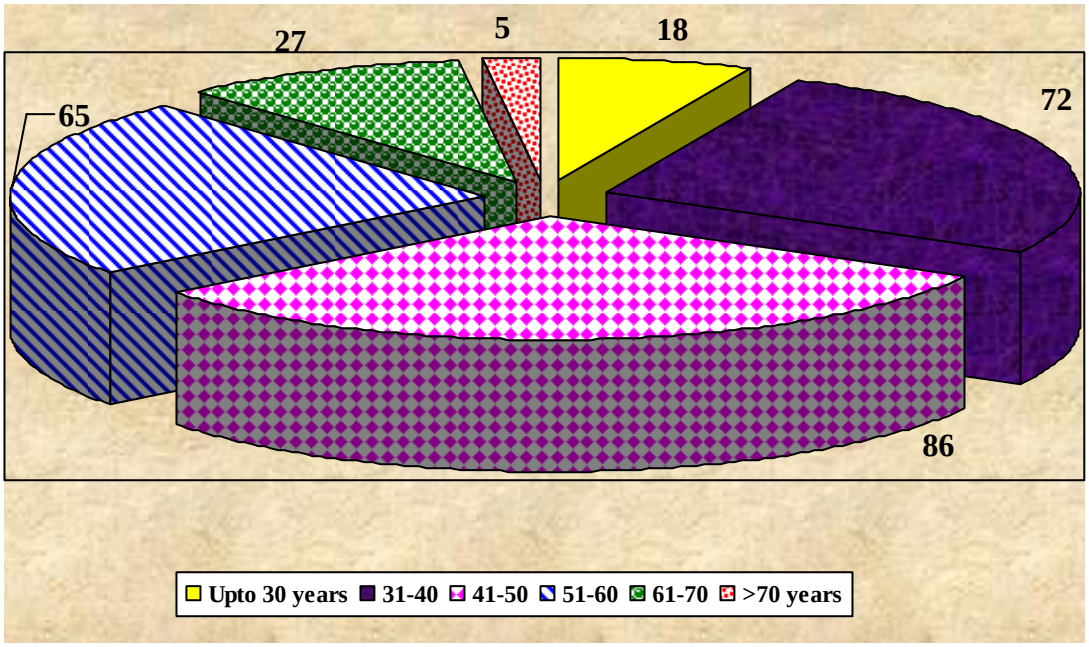
T4A(CHEST WALL FIXITY)

LABC AT ITS WORST !

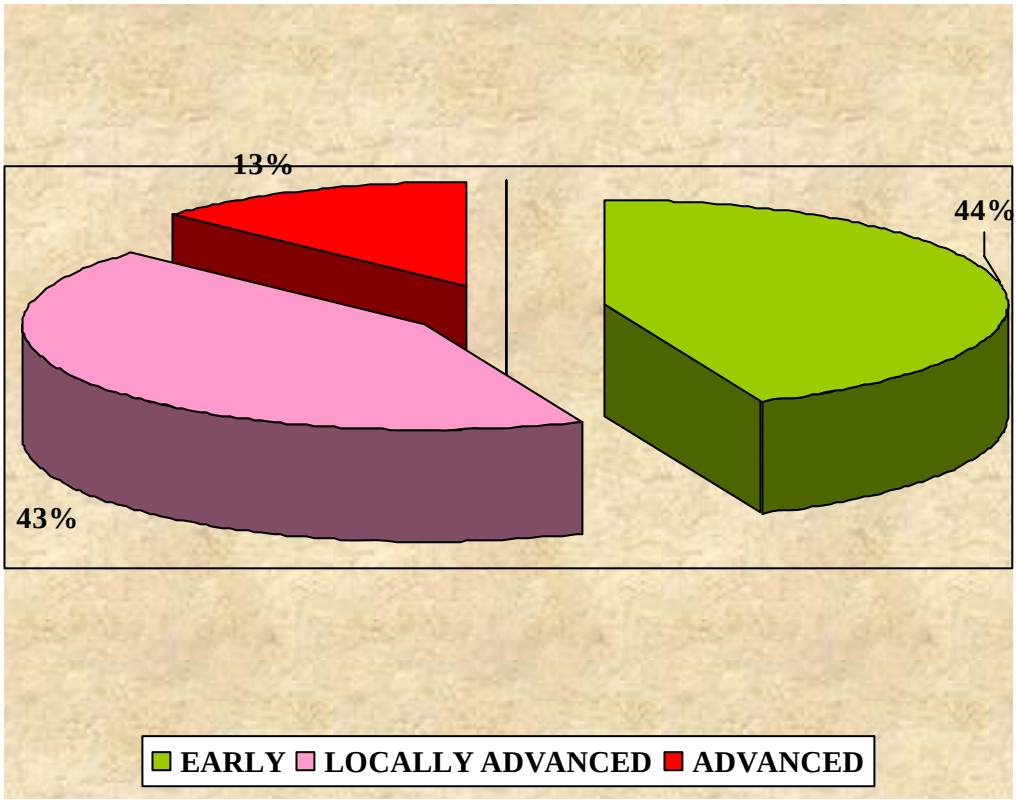
ULCERATIVE MALIGNANCY

NIPPLE DESTRUCTION

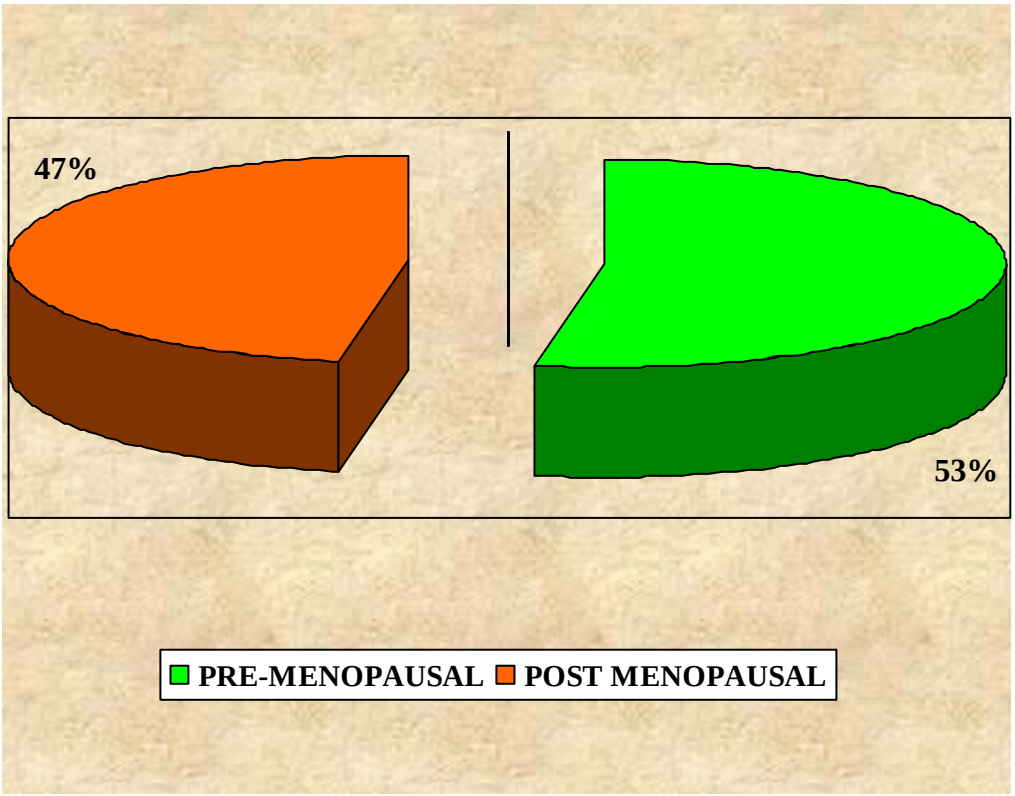
AGE DISTRIBUTION

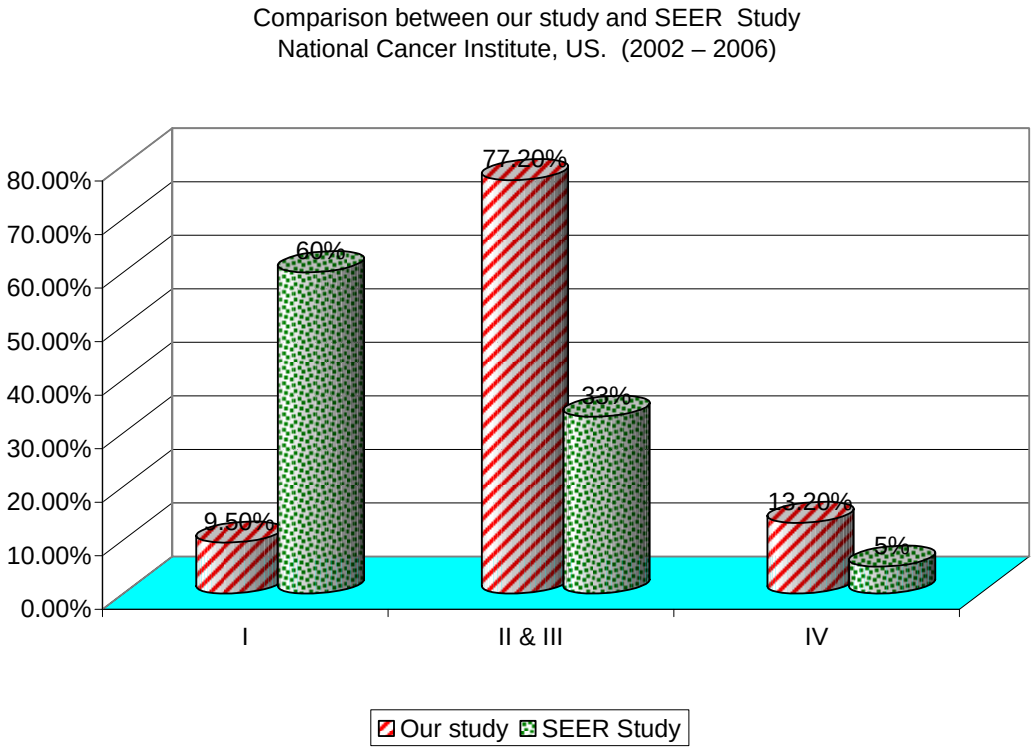


STAGE AT THE TIME OF PRESENTATION

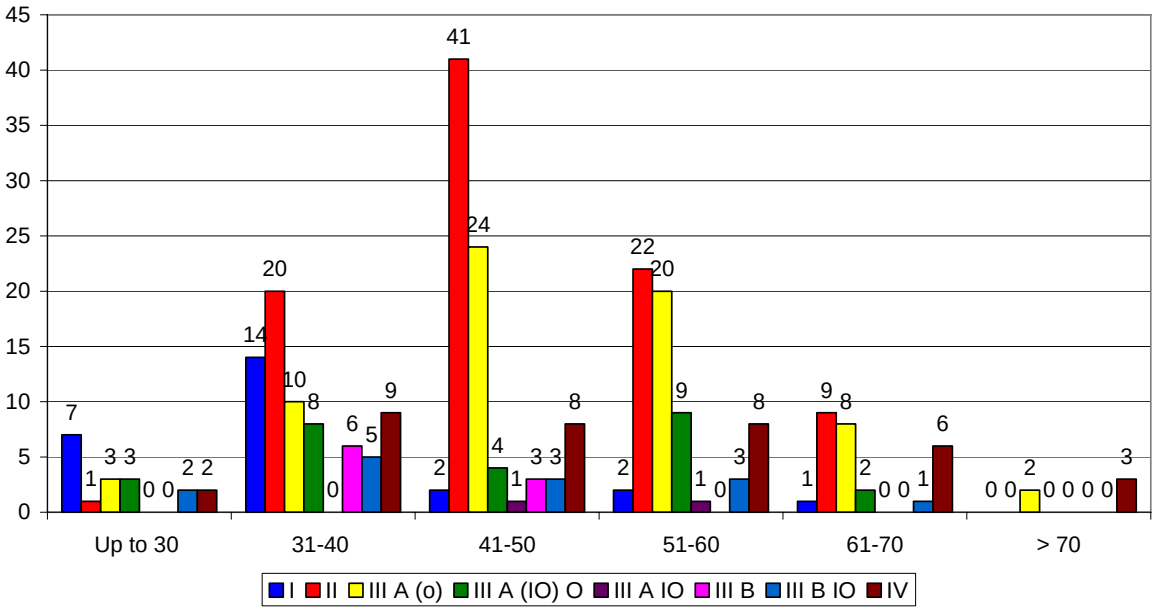


MENOPAUSAL STATUS

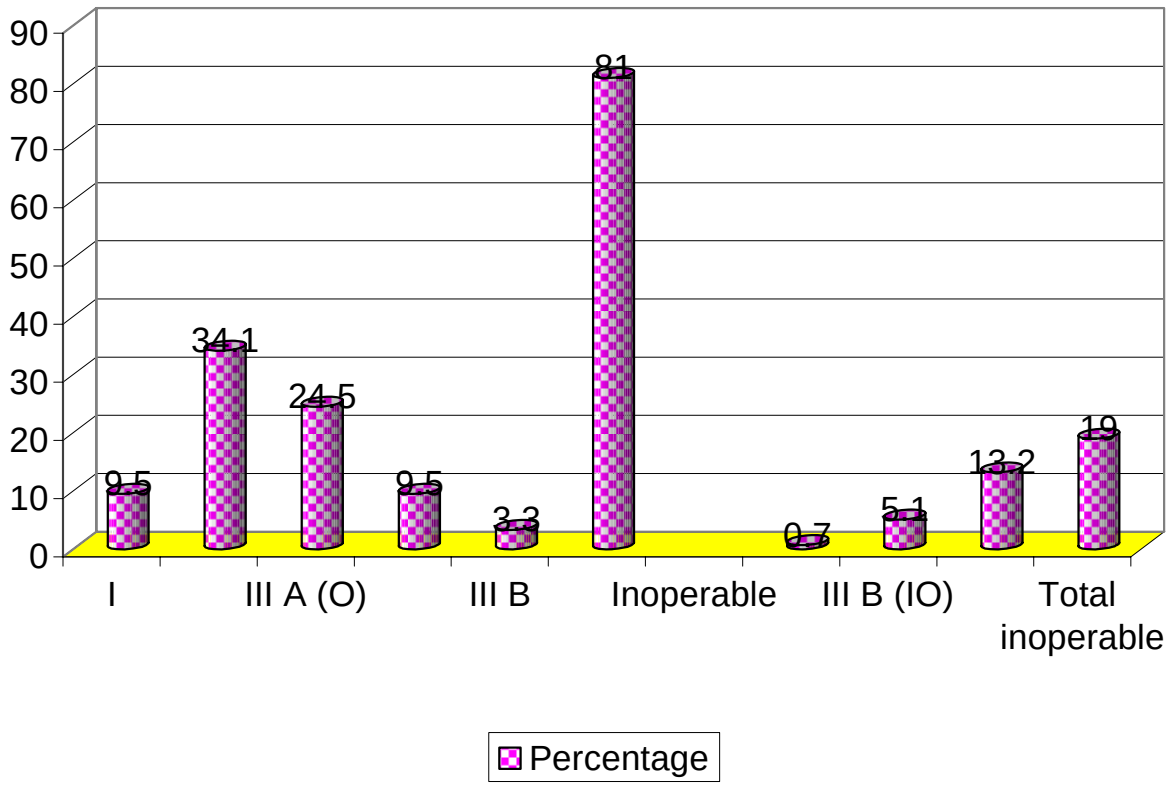



Comparison between our study and SEER Study
National Cancer Institute, US. (2002 – 2006)
80.00%
70.00%
60.00%
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
9.50%
60%
77.20%
33%
13.20%
5%
I
II & III
IV
Our study
SEER Study

AGE WISE DISTRIBUTION OF STAGING



INCIDENCE OF OPERABILITY



S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
1	RAJALAXMI	4	52	F	51028	R	$T_2 N_1 M_0$	A	1	II		2	MRM®	POST
2	POUN	3	45	F	54442	R	$T_{4b} N_2 M_1$	B	3	IV		8	Pal. Mast	PRE
3	FARITHA BAGAM	3	50	F	60721	L	$T_3 N_1 M_0$	A	2	III	a	3	MRM (L)	POST
4	PARVATHY	4	52	F	56985	L	$T_2 N_2 M_0$	A	1	II		2	MRM	POST
5	LAKSHMI	4	55	F	59489	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM neo-adj-chemo	POST
6	POORANAM	4	55	F	52450	B/L	$T_2 N_1$	A	1	II		2	B/L MRM	POST
7	MANJU	3	45	F	62285	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM	PRE
8	VIJAYALAXMI	3	44	F	63232	R	$T_1 N_1 M_0$	A	1	II		2	MRM	PRE
9	KAMATCHI	5	70	F	63246	L	$T_{4b} N_{3c} M_1$	B	3	IV		8	Pal.chemo	POST
10	SUNDARAM	2	40	F	62743	R	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
11	KATHAMMAL	5	70	F	66329	L	$T_{4b} N_2 M_1$	B	3	IV		8	Pal. Mast + chemo	POST
12	MEENATCHI	5	63	F	66207	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
13	VIJAYA	2	34	F	63454	L	$T_3 N_2 M_0$	A	2	III	B(IO)-->O	4	Pal. chemo	PRE
14	CHINNAMMAL	4	55	F	72558	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
15	GIRIJA	3	50	F	79016	R	$T_3 N_0 M_0$	A	1	II		2	MRM	POST
16	SUSEELA	3	50	F	80906	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM neo-adjchemo	POST
17	DHANAPACKIYAM	3	48	F	79297	R	$T_3 N_1 M_0$	A	2	III	a	3	MRM	POST
18	PONUTHAI	2	40	F	87406	R	$T_3 N_2 M_1$	B	3	IV		8	Pal. chemo +Hormona	PRE
19	GANGA	4	55	F	91199	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
20	PAPPAIYAMMAL	3	50	F	98320	R	$T_3 N_1 M_0$	A	2	III	a	3	MRM	POST
21	MARIAMMAL	1	27	F	97768	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
22	PANDIAMMAL	2	40	F	99127	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
23	PANCHAMMAL	4	58	F	28014	L	$T_{4b} N_2 M_1$	B	3	IV		8	Pal. Mast + chemo	POST
24	RAKKU	4	60	F	25749	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
25	SULOCHANA	5	62	F	33651	R	$T_3 N_0 M_0$	A	1	II		2	MRM	POST
26	CHANDRA	3	50	F	31232	R	$T_3 N_0 M_0$	A	1	II		2	MRM	POST
27	SUBBULAXMI	4	55	F	75129	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
28	PAPU BEGAM	3	45	F	77861	R	$T_4 N_2 M_0$	A	2	III	B(O)	6	MRM	PRE
29	KALIYAMMAL	4	55	F	77807	L	$T_2 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
30	RAMALAXMI	4	60	F	76989	R	$T_3 N_1 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
31	VIJAYALAXMI	4	60	F	73098	R	$T_x N_0 M_0$	A	1	I		1	MRM	POST
32	AMALAPUSPAM	3	47	F	74240	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM	PRE
33	LAKSHMI	3	50	F	72239	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
34	MUTHULAXMI	3	50	F	64106	R	$T_3 N_0 M_0$	A	1	II		2	MRM	POST
35	PAPPU	4	60	F	61426	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
36	MOOKAMMAL	3	43	F	55927	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
37	NAGALAXMI	3	42	F	51789	L	$T_1 N_1 M_0$	A	1	II		2	MRM	PRE
38	AROCKIAMMAL	4	60	F	649244	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
39	DHANAM	2	40	F	647069	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
40	ANNALAKSHMI	4	52	F	48102	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
41	POTTIAMMAL	2	40	F	46602	R	$T_4 N_2 M_0$	A	2	III	B(O)	6	MRM neo-adjchemo	PRE
42	PITCHAIAMMAL	4	51	F	31335	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
43	MUTHUMANICKAM	4	60	F	33832	L	$T_{4b} N_2 M_1$	B	3	IV		8	Pal. Mast + chemo RT	POST
44	SURIYAL BEGAM	2	36	F	31335	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM Chemo RT & hormona	PRE
45	PALANIAMMAL	4	55	F	30989	R	$T_{4b} N_{3c} M_0$	B	2	III	B(I)	7	Neo-adj-->pal.mast-->Chemo	POST
46	MURUGAMMAL	2	33	F	21752	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
47	ROOTH	3	50	F	20629	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM --->Chemo & RT	POST
48	PAPPA	4	60	F	5627	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
49	VALLI	4	58	F	90134	L	$T_2 N_0 M_0$	A	1	I		1	MRM --->Chemo	POST
50	SIKKANDER BEEVI	2	40	F	6614	R	$T_{4b} N_2 M_0$	A	2	III	B(O)	6	Neo-adj-Chemo-->MRM Chemo RT & hormona	PRE
51	GANDHI	2	36	F	6644	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM	PRE
52	AROCKIA SELVI	3	45	F	6928	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
53	SETHULAXMI	4	53	F	4352	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
54	MARI	4	55	F	105070	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
55	ARPUTHAM	4	60	F	100654	L	$T_{4b} N_2 M_0$	B	2	III	B(I)	7	Neo adj chmo -->NR --> Pal.chemo RT	POST

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
56	PARAMEESHWARI	4	56	F	99154	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --> adj chemo RT	POST
57	VASANTHA	2	40	F	98411	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
58	ARIVUKALANGIAM	3	43	F	98654	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM--> chemo RT	PRE
59	JEYALAKSHMI	2	34	F	93248	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
60	SOUNDARAM	4	60	F	85002	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM--> chemo RT	POST
61	LALITHA	1	27	F	90384	L	$T_x N_0 M_0$	A	1	I		1	MRM--> chemo	PRE
62	PARVATHI	3	50	F	86469	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
63	KALIAMMAL	3	50	F	80844	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
64	PITCHAIAMMAL	5	65	F	79616	R	$T_{4b} N_{3c} M_0$	B	2	II	B(I)	7	Neo adj chemo -->NR -->Pal. Mast	POST
65	ATHAYEE	3	48	F	84649	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
66	PIDARI	4	59	F	30867	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
67	KALIAMMAL	2	35	F	80502	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	PRE
68	VALLI	3	45	F	77714	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
69	CHINNIAMMAL	2	37	F	77613	R	$T_1 N_1 M_0$	A	1	II		2	MRM	PRE
70	KANNIAMMAL	5	70	F	75811	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
71	REVATHY	4	55	F	76916	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
72	RAIYA BANU	2	32	F	70011	R	$T_{4b} N_2 M_0$	A	2	III	B(O)	6	Neo adj chemo -->MRM --> chemo +RT	PRE
73	LAKSHMI	4	57	F	70111	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
74	ANTHONIAMMAL	2	40	F	66352	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
75	VASANTHI	2	33	F	67825	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
76	JAYAKODI	3	45	F	71462	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo+RT+hormona	PRE
77	RAJAMMAL	5	63	F	68889	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
78	VASANTHA	2	33	F	67825	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo+RT+hormona	PRE
79	DHANUSKODI	2	40	F	65740	L	$T_{4b} N_2 M_0$	A	2	III	B(O)	6	Neo adj chemo-->MRM-->C+RT	PRE
80	MURUGESHWARI	1	30	F	63675	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo+RT+hormona	PRE
81	JEBALKUMARI HELENE	3	48	F	56849	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
82	PARVATHY	5	70	F	55507	R	$T_{4b} N_{3c} M_1$	B	3	IV		8	Pal.mast-->Chemo	POST

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
83	KUPPAMMAL	3	45	F	47810	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
84	NAGOMI	5	70	F	54770	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo	POST
85	INDRA	2	40	F	53430	L	$T_{4c} N_2 M_0$	A	2	III	B(O)	6	Neo adj chmo --> MRM -->chemo RT	PRE
86	VALLI	3	47	F	45422	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
87	SHANDHI	3	44	F	48896	R	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
88	PARVATHY]	6	75	F	47803	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT	POST
89	CHILLAIAMMAL	3	43	F	41797	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
90	MUTHULAKSHMI'	3	50	F	45494	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT	POST
91	AMINA	2	40	F	45495	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
92	RENGANAYAKI	4	58	F	47933	R	$T_3 N_0 M_0$	A	1	II		2	MRM	POST
93	ESWARI	2	40	F	46817	R	$T_{4b} N_2 M_0$	A	2	III	B(O)	6	Neo adj chemo -->MRM --> chemo +RT+ hormona	PRE
94	KAMATCHI	4	55	F	41147	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
95	SHIVANAMMAL	3	50	F	38135	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
96	JEYARANI	3	45	F	38173	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	PRE
97	KUPPAMMAL	3	50	F	36731	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
98	MEENAL	3	50	F	29434	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
99	SUBBUTHAI	2	38	F	33283	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
100	SUSEELA	4	52	F	34288	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
101	CHITHRA	2	31	F	33053	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
102	INDRANI	3	48	F	26532	R	$T_{4b} N_1 M_0$	A	2	III	B(O)	6	Neo adj chemo -->MRM --> chemo +RT+ hormona	PRE
103	MUNIAMMAL	3	50	F	29802	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
104	SELVI	1	29	F	27274	R	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
105	BAMA	2	35	F	29069	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	PRE
106	VELANKANNI	2	37	F	28373	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	PRE
107	PALANIAMMAL	4	60	F	29171	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
108	DHANALAKSHMI	6	78	F	24517	L	$T_{4b} N_2 M_1$	B	3	IV		8	Pal.mast-->Chemo	POST
109	JOTHIAMMAL	2	40	F	95259	L	$T_2 N_1 M_0$	A	1	II		2	Pal.mast-->Chemo	PRE

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
110	PITCHAIMANI	3	41	F	97298	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	PRE
111	SAROJA	3	47	F	100889	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
112	USHA	2	35	F	20465	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
113	LAXMI	5	68	F	20599	R	$T_{4b} N_2 M_1$	B	3	IV		8	Pal.mast-->Chemo	POST
114	RAMU	4	58	F	91096	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
115	SUNDARI	4	60	F	101668	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	POST
116	KAMALA	3	50	F	100896	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
117	POONGOTHAI	3	45	F	10344	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
118	PERIYAKKAL	3	48	F	90171	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	PRE
119	JOTHI	4	59	F	90190	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	POST
120	PALANIAMMAL	3	45	F	91117	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
121	VIJAYA	4	51	F	36872	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
122	GANTHIMATHI	5	65	F	70792	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
123	RAJAMMAL	3	42	F	76611	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
124	VALLIAMMAL	5	66	F	76735	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
125	MUTHUMARI	3	50	F	76353	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
126	KUMUDHAVALLI	1	26	F	76571	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
127	AARAIJEE	3	45	F	76577	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	PRE
128	RAKKAMMAL	3	42	F	76590	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
129	PITCHAIAMMAL	2	40	F	78860	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
130	ANDAMMAL	3	45	F	78896	L	$T_3 N_{3C} M_0$	A	2	III	B(IO)	3	PALLIATIVE chemo RT	PRE
131	RASAMMAL	2	40	F	84941	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
132	ASNAMMAL	3	47	F	85743	B/L	$T_{4c} N_2 M_1$	B	3	IV		8	PALLIATIVE chemo RT	POST
133	RANJITHAM	4	55	F	83877	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
134	SHANTHI	4	52	F	89006	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	POST
135	CHITHRAJEE	5	65	F	87379	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	POST
136	KALAVATHY	2	32	F	87681	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
137	LAXMI	4	55	F	95630	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	POST
138	SURYAGANDHI	5	65	F	97537	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
139	KUPPAMUTHU	2	40	F	610814	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
140	KOWSALYA	3	50	F	20264	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
141	RANI	3	42	F	101443	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
142	ARASAMMAL	2	40	F	25341	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	PRE
143	LAXMI PALANIYACHI	3	45	F	29515	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
144	SANHARI	3	50	F	30734	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT+ hormona	POST
145	MANIAMMAL	4	58	F	30829	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
146	SAVITHRI	4	60	F	25325	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
147	MAHABOOBA	3	44	F	32337	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
148	MARUDHAYEE	4	60	F	33522	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT	POST
149	AYYAMMAL	3	50	F	32138	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT	POST
150	VELLAIAMMAL	3	45	F	32398	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
151	PANDIAMMAL	4	60	F	37725	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT	POST
152	ARASAMMAL	3	49	F	36862	B/L	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
153	IRULAYEE	6	75	F	42416	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM --chemo RT	POST
154	SOWDAMANI	5	69	F	39453	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
155	JATHIMUTHU	2	35	F	42117	R	$T_x N_1 M_0$	A	1	I		1	MRM	PRE
156	GANDHIMATHI	1	30	F	45387	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
157	PANCHAVARNAM	2	40	F	46765	L	$T_{4c} N_2 M_1$	B	3	IV		8	Pal.chemo + hormona	PRE
158	AMUTHARANI	2	35	F	49721	L	$T_{4b} N_{3c} M_1$	B	3	IV		8	Pal.mast-->Chemo	PRE
159	SARASWATHY	4	52	F	48112	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
160	MARIAMMAL	4	55	F	49620	L	$T_{4c} N_{3c} M_1$	B	3	IV		8	Pal.chemo	POST
161	SHERINA BANU	3	48	F	52133	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
162	KAMARNISHA	3	45	F	49599	L	$T_3 N_1 M_0$	A	2	III	A	3	Pal.chemo RT+ hormona	PRE
163	VIJIYA	3	50	F	50917	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
164	MUMTAJ	3	45	F	49099	L	$T_{4b} N_{3c} M_0$	B	2	III	B(IO)	7	Neo adj chemo -->NR -->Pal. Mast+chemo RT	PRE

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
165	KRISHNAMMAL	5	65	F	56910	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
166	MUTHUPILLAI	5	70	F	58906	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
167	SIVABACKIAM	4	55	F	37689	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
168	FATHIMA	2	38	F	63450	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
169	VASANTHI	4	55	F	59461	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
170	SARASWATHY	3	41	F	66434	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
171	RANI	2	40	F	70671	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	PRE
172	GOMU	3	42	F	69648	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
173	FATHIMABEEVI	3	45	F	72314	R	$T_{4b} N_{3c} M_0$	B	2	III	B(I)	7	Neo adj chemo -->NR -->Pal. Mast+chemo RT	PRE
174	VIJAYALAXMI	4	56	F	74199	L	$T_{4b} N_2 M_1$	B	3	IV		8	Pal.mast-->Chemo	POST
175	POOHUMAYIL	2	40	F	70357	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
176	JAMEELA	2	37	F	72800	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
177	PARVATHY	3	45	F	78204	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
178	VELAMMAL	4	58	F	80237	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
179	REKHA	1	30	F	71086	R	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
180	CHITHRADEVI	1	28	F	79969	R	$T_{4b} N_2 M_0$	B	2	III	B(I)	7	Neo adj chemo -->NR -->proueceboe Pal. Mast	PRE
181	DHANALAKSHMI	2	37	F	82857	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
182	MUTHULAXMI	4	57	F	82836	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
183	SIVAKAMI	4	52	F	83951	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
184	SOUNDARAM	2	40	F	85002	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
185	INDRA	2	37	F	89260	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
186	THILAGAVATHI	3	49	F	92087	L	$T_{4b} N_3 M_0$	B	2	III	B(IO)	7	Neo adj chemo -->NR -->Pal. Mast	PRE
187	PANCHU	2	37	F	92090	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
188	PITCHAIAMMAL	3	48	F	92007	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
189	TAMILSELVI	2	40	F	97916	R	$T_{4b} N_{3c} M_0$	B	2	III	B(I)	7	Neo adj chemo -->NR -->Pal. Mast	PRE
190	MEENAKSHI	2	38	F	94783	L	$T_{4b} N_2 M_0$	B	2	III	B(I)	7	Neo adj chemo -->NR -->Pal. Mast	PRE
191	LAKSHMI	1	25	F	10147	R	$T_x N_0 M_0$	A	1	I		1	MRM	PRE

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
192	BOOMA	2	35	F	103831	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
193	JAYANTHI	3	42	F	106681	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
194	SYED ROHIA	1	29	F	105675	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
195	SHANTHI	2	36	F	105361	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
196	USHARANI	2	36	F	8796	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
197	RAKKAMMAL	2	40	F	9052	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT+Hormonal	PRE
198	SULTHANOMMAL	3	50	F	97184	R	$T_{4c} N_{3c} M_0$	B	3	IV		8	pal. chemo	POST
199	EDANI	5	65	F	20986	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
200	PONMUDI	5	70	F	20901	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
201	PUSPAM	2	40	F	24631	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
202	CHINNAPONNU	2	35	F	26453	R	$T_{4b} N_{3c} M_1$	B	3	IV		8	Pal.mast-->Chemo+hormonal	PRE
203	NAGAMMAL	4	60	F	23613	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
204	LAKSHMI	5	70	F	26790	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
205	THEIVANAI	2	35	F	26564	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
206	MUTHUPILLAI	3	50	F	33478	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
207	KARUPAYEE	4	60	F	30395	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
208	VELAMMAL	4	60	F	35186	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
209	JEBAKANI	2	35	F	36199	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
210	SUDHA	1	30	F	34433	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE
211	VAIRAKKAL	5	65	F	34204	R	$T_2 N_1 M_0$	A	1	I		1	MRM	PRE
212	MALLIGA	4	56	F	20091	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
213	BOOMADEVI	3	45	F	4252	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
214	INDIRA	3	45	F	47671	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	PRE
215	SARASWATHY	2	32	F	47762	R	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
216	GURUVAMMAL	3	43	F	56422	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
217	PANDIAMMAL	1	30	F	49719	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
218	RATHINAMMAL	5	65	F	54861	L	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
219	SUSEELA	2	39	F	57660	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	PRE

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
220	KALIAMMAL	4	57	F	60891	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
221	SORNAMMAL	3	43	F	68027	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
222	TAMILSELVI	2	39	F	75288	R	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
223	NAGAMMAL	4	60	F	73641	L	$T_3 N_2 M_0$	B	2	III	A(IO)	5	MRM	POST
224	PALANIAMMAL	2	40	F	64447	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
225	MEHIRA BEGAM	2	32	F	67858	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	PRE
226	NEELA	2	40	F	65452	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
227	SELVI	2	38	F	70287	R	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
228	PADMAVATHI	5	65	F	62379	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
229	VELLIYAMMAL	5	63	F	67500	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
230	ALAGI	3	50	F	69146	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	PRE
231	MARIAMMAL	5	62	F	66648	R	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
232	MALLIGA	3	50	F	68989	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	POST
233	AMEENA BEEVI	3	50	F	66675	L	$T_2 N_1 M_0$	A	1	II		2	MRM	POST
234	MAYAMMAL	3	50	F	70312	L	$T_3 N_2 M_0$	B	2	III	A(IO)	5	MRM	POST
235	KRISHNAMMAL	4	60	F	68021	R	$T_3 N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
236	AMARAVATHY	4	60	F	69975	R	$T_3 N_1 M_0$	A	2	III	A	3	MRM	POST
237	SIVAKAMI	2	40	F	69962	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
238	KUPPATHAL	3	46	F	32106	R	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
239	JOTHI	1	30	F	36222	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
240	MAHESWARI	1	29	F	3819	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT+Hormonal	PRE
241	MARUTHU	3	50	F	45580	L	$T_x N_0 M_0$	A	1	I		1	MRM	POST
242	JHANSI RANI	3	46	F	40102	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT	PRE
243	SHENBAGAVALLI	4	55	F	60084	R	$T N_2 M_0$	A	2	III	A(IO)-->O	4	MRM	POST
244	VALLIAMMAL	3	47	F	59885	L	$T_2 N_1 M_0$	A	1	II		2	MRM	PRE
245	SUBBULAXMI	1	30	F	666737	L	$T_3 N_1 M_0$	A	2	III	A	3	MRM -->chemo RT+Hormonal	PRE
246	ROOPA	2	34	F	77892	L	$T_x N_0 M_0$	A	1	I		1	MRM	PRE
247	MOOKAYEE	2	40	F	416	L	$T_3 N_{3c} M_1$	B	3	IV		8	Pal.chemo RT+Hormonal	PRE

S.No	Name	AGE GROUP	Age	Sex	IP No	Side	TNM Stage	O/IO	TYPE OF BC	Staging		STAGE	Treatment given	Menopausal status
248	RASIYABANU	2	32	F	1880	R	$T_{4b}\ N_3\ M_0$	B	2	III	B(IO)	7	Pal.chemo	PRE
249	GOMATHY	2	40	F	4613	R	$T_{4c}\ N_3\ M_0$	B	2	III	B(IO)	7	Pal.chemo	PRE
250	PARVATHY	6	75	F	6950	R	$T_{4c}\ N_2\ M_1$	B	3	IV		8	Palliative chemo	POST
251	PANJAVARNAM	2	40	F	8569	L	$T_2\ N_1\ M_0$	A	1	II		2	MRM	PRE
252	RASIYA	4	60	F	9947	L	$T_{4b}\ N_{3c}\ M_1$	B	3	IV		8	Palliative chemo	POST
253	ARUVAGAM	1	30	F	24044	L	$T_{4c}\ N_2\ M_0$	B	2	III	B(I)	7	Pal.chemo	PRE
254	SAVITHRI	6	74	F	26446	L	$T_{4b}\ N_{3c}\ M_1$	B	3	IV		8	Palliative	POST
255	KADHAR BEEVI	2	40	F	31683	L	$T_{4b}\ N_{3c}\ M_0$	B	2	III	B(I)	7	palliative chemo	PRE
256	IRULAYEE	1	30	F	33641	L	$T_{4b}\ N_2\ M_1$	B	3	IV		8	palliative chemo	PRE
257	ALAGI	4	55	F	41454	R	$T_{4b}\ N_{3c}\ M_0$	B	2	III	B(IO)	7	Palliative chemo	POST
258	THEDASELVAM	3	47	F	44025	L	$T_{4b}\ N_2\ M_0$	A	2	III	B(O)	6	Neo adj chemo RT --> MRM --> chemo RT	PRE
259	PANCHAVARNAM	2	35	F	55153	L	$T_{4b}\ N_2\ M_0$	B	3	IV		8	Toilet. mast	PRE
260	GOMATHY	2	40	F	20407	L	$T_{4b}\ N_{3c}\ M_1$	B	3	IV		8	Toilet. mast	PRE
261	DEIVANAI	5	63	F	46247	R	$T_{4b}\ N_{3c}\ M_1$	B	3	IV		8	Toilet. mast	POST
262	ANNAMALAIAMMAL	5	70	F	44331	R	$T_{4b}\ N_2\ M_1$	B	3	IV		8	Toilet. mast	POST
263	PALANIAMMAL	3	50	F	33253	R	$T_{4b}\ N_2\ M_1$	B	3	IV		8	Pal.mast	POST
264	GNANAMANI	4	60	F	99099	R	$T_{4b}\ N_2\ M_1$	B	3	IV		8	Pal.mast	POST
265	ALAGI	1	21	F	50800	R	$T_{4b}\ N_2\ M_1$	B	3	IV		8	Pal.chemo	PRE
266	SARASWATHY	3	50	F	78176	L	$T_{4c}\ N_2\ M_1$	B	3	IV		8	Pal.chemo	POST
267	KARUPAYEE	4	60	F	107148	R	$T_{4b}\ N_{3c}\ M_1$	B	3	IV		8	Pal.chemo	POST
268	KANNAKI	3	43	F	102935	R	$T_{4b}\ N_2\ M_1$	B	3	IV		8	Pal.chemo	PRE
269	SURYAGANDHI	3	50	F	101604	L	$T_{4c}\ N_2\ M_1$	B	3	IV		8	Pal.chemo	POST
270	MOOKAYEE	2	40	F	98090	L	$T_{4b}\ N_{3c}\ M_1$	B	3	IV		8	Pal.chemo	PRE
271	ARAYAMMAL	2	40	F	96735	R	$T_3\ N_{3c}\ M_1$	B	3	IV		8	Pal.chemo	PRE
272	INDRANI	3	48	F	86755	L	$T_2\ N_2\ M_1$	B	3	IV		8	Pal.chemo	PRE
273	MARIAMMAL	4	60	F	72301	L	$T_3\ N_{3c}\ M_1$	B	3	IV		8	Pal.chemo	POST

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PROFORMA

Name : IP No. :

Age : Address :

Sex : Occupation :

DOA : DOD :

Presentation :

Complaints :

Lump in the breast

Nipple discharge

Nipple retraction

Pain

Ulcer / nodule

Swelling in the axilla

Cough dyspnoea, chest pain

Bony pain

Anorexia, jaundice

Loss of weight

Past History :

H/o previous surgery in the breast

H/o OCPs intake and HRT intake

Personal H: Diet **O/E Anaemia**

Age at Menarche PR -

Menstrual H : BP -

Age at marriage : CVS -

Age at 1st child birth : RS -

No. of Children :

Duration of breast feeding:

Family History :

Examination :

Size, nipple retraction, nipple discharge, peau – d- orange,

Ulcer / nodule

Fixity to underlying structures

Nodal status

Bony tenderness

Hepatomegaly

Ascites

Pleural effusion

Diagnosis and Stage :

Investigation :

Biopsy

Chest X ray

USG abdomen

Bone Scan

Management :

Surgery

Chemo → Neoadjuvant → Response

Chemo → Adjuvant

RT → Neo adjuvant → Response

RT → Adjuvant